STOCHASTIC MODELS IN GENETICS

DISSERTATION SUBMITTED
IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE AWARD OF THE DEGREE OF

Master of Philosophy

IN

STATISTICS

BY

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ALIGARH (INDIA)
1990

"STOCHASTIC MODELS IN GENETICS"

P R E F A C E

The present dissertation entitled "STOCHASTIC MODELS IN GENETICS" gives a brief description of the Stochastic Models and their applications to Genetics. Though applied in nature the Stochastic Models arising in such disciplines as Genetics etc are of immense theoretical interest. Scientists are, therefore, actively engaged in investigating both these aspects thereby enriching them theoretically and at the same time windening the scope of their applications to the real life situations. It is practically impossible to prsent a review on all that has been done in this area and therefore an humble effort has been made to identify some of the problems that has been considered during the past few years and to review the relevant contributions that have been made so far. The dissertation consists of four chapters.

Chapter I is essentially introductory in nature and contains the preliminary ideas from the theory of Stochastic Processes which are relevant to the subsequent discussions.

The Chapter II describes some Stochastic Models applied to POPULATION GROWTH. Some recent contributions suggesting improvements in the classical models have been included.

Chapter III presents a brief discussion on "ASSORTATIVE MODELS" In fact this area has great potential and promise for further research. This is evident from the quantum of work that is being done presently. The nature of problems that are of interest can

be judged from the papers that have been reviewed in the Chapter.

In the Chapter IV some contributions are made in an area of "PREDATOR - PREY MODELS" which is again an asset for further research.

In the end, a comprehensive bibliography of the various publications that have been referred to is given in the present DISSERTATION.

I feel great pleasure in taking this opportunity to acknowledge my deep sense of gratitude to Dr S Rehman, Professor, Department of Statistics, Aligarh Muslim University, Aligarh, for his most valuable and precious guidance.

I have to express my sense of gratitude to all my teachers, friends and colleagues for their cooperation.

Finally, while closing my 'PREFACE'. I cannot forget my parents for their timely, valuable and precious guidance which made it possible for me to bring this MANUSCRIPT to the final stage.

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MAY 1990

C O N T E N T S

CHAPTER-I

SECTION "A":

PRELIMINARY IDEAS FROM THE THEORY OF STOCHASTIC PROCESSES

1.1 INTRODUCTION: A Stochastic Process is the mathematical abstraction of an empirical process whose development is governed by probabilistic laws. Since the last two decades it has been realized that probability models are more realistic than deterministic models in solving many problems. Since the work of Kolmogorov (1931) and Fellor (1936), the theory of Stochastic Processes has developed rapidly. The systematic treatment of the subjects are given by Chiang (1961), Doob (1953), Fellor (1957, 1966), Harris (1963), Cox and Miller (1965), S Karlin (1966), Kendall (1948), Parzan (1962) etc.

The theory of Stochastic Processes is now finding increasing applications to problems in physical sciences, which deal with processes involving some random elements in their structure. During past two decades the tendency to use the results of stochastic theory in developing the models in applied science and engineering has increased considerably. And this factor helped very much in the development of stochastic theory itself. Sometimes it seems to be necessary to find or apply new moethods or techniques for the formulation and solution of problems occuring in the realm of natural sciences. In recent years due to increasing applications the need for new methods and techniques had been very keenly felt.

1.2 STOCHASTIC PROCESSES:

DESCRIPTION AND DEFINITIONS:

Suppose Ω is a sample space and 'P' a probability measure.

DEFINITION: A random variable 'X' with values in the set 'E' is a function which assigns a value X(W) in E to each outcome W in Ω.

Usual examples of E are the set of non-negative integers N = [o,1,2,....,], the set of all integers [..-2,-1,o,1,2,...], the set of all real numbers R= (-oo,+oo) and the set of all non-negative real numbers R_+ =[o,oo). In the first two cases and more generally when E is finite or countably infinite X is said to be a discrete random variable.

Otherwise if E is infinite as in the last two cases of R and R_+, then X is said to be continuous random variable.

DEFINITION: A stochastic process with state space E is a collection [$X_{(t)}$, T of random variable $X_{(t)}$ defined on the same probability space and taking values in E. The set T is called its parameter set or index set. If T is countable, especially if T = N = [o,1,2,...], the process is said to be discrete parameter process. Otherwise if T is not countable, the process is said to have a countinuous parameter. In the latter case the usual examples are T = R_+ = [o,oo) and T= [a,b] $\subset$ R=(oo,oo). It is customery to think of the index 't' as representing time, and then one thinks of X_t as 'state' or the 'position' of the process at time 't'. The set E is accordingly called the state space of the process.

In indentifying the nature of stochastic processes, as a first step, it helps to classify them on the basis of the nature of their parameter space and state space. Accordingly we have

(a) Stochastic Processes with discrete parameter and discrete state space : e.g.

(a_1) Consumer preferences observed on a monthly basis

(a_2) The number of defective items in an acceptance sample scheme. The number of item inspected is the indexing parameter

(b) Stochastic Processes with continuous parameter space and descrete state space, e.g.

(b_1) Amount of inventory on hand (when they are discrete items) over a period of time

(b_2) Number of students waiting for the bus at any time of the day

(c) Stochastic Processes with discrete parameter space and continuous state space, e.g.

(c_1) Suppose the production is not denumerable (such as cloth, gasoline etc.) and the inventory on hand is observed only at discrete epochs of time. Then we have a stochastic process belonging to this class

(d) Stochastic Processes with continuous parameter space and state space, e.g.

(d_1) The content of a dam observed over an interval of time ,

(d_2) The waiting time of an arriving job until it gets into service. The arriving time of the job is now the parameter.

DESCRIBING THE PROBABILITY LAW OF S.P: For every $t_1, t_2, \ldots t_n$ in T, the corresponding random variable $X(t_1)$, $X(t_2)$,..., $x(t_n)$ have the joint n-dimentional dist^n function

$$F_n(x_1, x_2, x_3, \ldots, x_n; t_1, t_2, \ldots, t_n)$$
$$= P\left[X(t_1) \leq x_1, X(t_2) \leq x_2, \ldots, X(t_n) \leq x_n\right] \ldots (1.2.1)$$

The family of all these joint probability distributions for n = 1,2,.... and all possible values of the t_j' s constitute the family of finite demensional distribution associated with X(t) - process. Thus in order to describe the probability law of a S.P [X(t), t ∈ T] we must specify the joint probability law of the n random variables $X(t_1)$, $X(t_2)$,...., $X(t_n)$ for all integers n and n points $t_1, t_2, \ldots, t_n$ in T. To specify the joint probability law we must specify either (i) the joint distribution function given by (1.2.1) above or (ii), the joint characteristic function given for all real numbers u_1, u_2,....,u_n by

$$\phi_{X(t_1), \ldots, X(t_n)}(u_1, u_2, \ldots, u_n) = E\left[\exp i(u_1 X(t_1) + \ldots + u_n X(t_n)\right]$$
$$= \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \exp\left[i(u_1 x_1 + \ldots + u_n x_n\right] dF(x_1, \ldots, x_n) \ldots (1.2.2)$$

It is to be noted here that the family of finite dimensional distributions associated with the X(t) - process must satisfy the following two conditions which are called Kolmogorov consistency conditions:

1. (SYMMETRY):

The symmetry condition requires that the n-dimensional distribution function F should be symmetric in all pairs (x_j, t_j) so that F should remain invariant when x_j and t_j are subjected to same permutations.

2. (CONSISTENCY):

The consistency condition is expressed by the relation

$$\lim_{x_n \to \infty} F_n(x_1, x_2, \ldots\ldots, x_n; t_1, t_2, \ldots\ldots, t_n)$$

$$= F_{n-1}(x_1, x_2, \ldots, x_{n-1}; t_1 \ldots, t_{n-1}) \ldots (1.2.3)$$

CLASSIFICATION OF STOCHASTIC PROCESSES:

The mean elements distinguishing stochastic processes are in the nature of the space E, the index parmeter T, and the dependence relation among the random variable X_t.

As defined earlier the state space of a stochastic process is the space in which possible values of each X_t lie. If E = (o,1,2...) the stochastic process is referred to as integer valued or a discrete state space process. If E = the real line $(-\infty, \infty)$, we call X_t a real valued stochastic process. If E is Euclidean K space then X_t is said to be a K-vector process.

We now describe some of the classical types of stochastic processes, characterized by different dependence relation among X_t. Unless stated contrary we take T= $[o, \infty]$ and assure that random variables X_t are real valued.

(a) PROCESSES WITH STATIONARY INDEPENDENT INCREMENTS:

If the random variables

$X_{t_2}-X_{t_1}, X_{t_3}-X_{t_2}, \ldots . X_{t_n} - X_{t_{n-1}}$ are independent, for all choices of $t_1, t_2, \ldots, t_n$ satisfying

$t_1 < t_2 < \ldots . < t_n$

then we say that X_t is a process with independent increments. If the index set contains a smaller index t_o, it is also assumed that

$X_{to}, X_{t_1}-X_{to}, \ldots . , X_{tn}-X_{tn-1}$ are independent. If the index set is discrete i.e. T=(o,1,....)then a process with independent increments reduces to a sequence of independent random variables $z_{o=X_o}, z_i=X_i - X_{i-1}$, (i=1,2....) in the sense that proving the individual distributions of $Z_o, Z_1, \ldots,$ we can determine the joint distribution of any finite set of the X_i. In fact

$X_i=Z_o+Z_1+\ldots . . .+Z_i$, i=o,1,2,..

If the distribution of the increments $X_{t+h}-X_t$ depends only on the length h of the interval and not on the time t, then the process is said to have stationary increments. For a process with stationary increments the distribution of $X_{t_1+h}-X_{t_1}$ is the same as the distribution of $X_{t_2+h} - X_{t_2}$, no matter what the values of t_1, t_2 and h.

(b) STATIONARY PROCESSES: A stochastic process is stationary if its finite dimensional distributions

are invariant under arbitrary translation of the time parameter. Thus the process X(t) is stationary for all $t_1, t_2, \ldots t_n$ and $h > 0$ if for all n

$$P[X(t_1) \leq x_1, \ldots, X(t_n) \leq x_n]$$
$$= P[X(t_1+h) \leq x_1, \ldots X(t_n+h) \leq x_n]$$
$$t_i \in T, (t_i+h) \in T, \quad i = 1,2,\ldots,n \ldots (1.2.4)$$

such processes are sometimes said to be strictly stationary. A process is called stationary to order n if the equation (1.2.3) holds for some particular n. rocesses which are not stationary are called non-stationary or evolutionary processes.

A stochastic process is said to be vide sense stationary or covariance stationary if it posseses finite second moments and if

$$Cov(X_t, X_{t+h}) = E(X_t, X_{t+h}) - E(X_t)E(X_{t+h}),$$

depends only on h for all $t \in T$. A stationary process that has finite second moment is covariance stationary. However, there are covariance stationary processes that are not stationary.

(c) GAUSSIAN OR NORMAL PROCESS:
If the joint distribution functions are multivariate normal, the process is called Gaussian or Normal.

(d) MARKOV PROCESSES: A stochastic process is said to be Markovian if given the values of X(t), the value of X(s)
for $s < t$ does not depend on the value of x(u), $u < t$, in other words, the future behaviour of the process depends only on the present state not on the past. Formally, a process is said to be a Markov process if

$$P(a < X_t \leq h / X_{t_1} = x_1, X_{t_2} = x_2, \ldots, X_{t_n} = x_n)$$
$$= P(a < X_t \leq h / X_{t_n} = x_n) \ldots\ldots\ldots (1.2.5)$$

where $t_1 < t_2 < t_3 < \cdots\cdots < t_n < t$.

Let A be an interval on the real line. The limit function

$$P(x, s; t, A) = P(x_t \in A / x_s = x),\ t < s \quad \ldots (1.2.6)$$

is called the transition probability function and is basic to the study of the structures of Markov processes. We may express the condition (1.2.5) as follows:

$$P[a < X_t \leq b / X_{t_1} = x_1 \cdots X_{t_n} = x_n] = P(x_n, t_n; t, A) \quad \ldots\ldots\ldots (1.2.7)$$

where $A = (a < X_i \leq b)$.

A Markov process is said to have stationary transition probability or to be homogeneous in time if p(X,s; t,A) defined in (1.2.6) is a function of (t-S) only, since the probability P(X,s;t,A) is a conditional probability given the present state, a Markov process with stationary transition probability cannot be a stationary process.

A Markov process having a finite or denumerable state space is called a Markov Chain. Thus we have descrete time Markov Chains as well as continuous time Markov Chains.

1.3 MARKOV CHAINS: A discrete time Markov Chain $[X_n]$ is a Markov Stochastic Process whose state space is a countable or finite set and for which T= (o,1,2,.....). The value X_n is referred to as the outcome of the n^{th} trial. For convenience we level the state space by the non-negative integers (o,1,2,.... and we speak of X_n being in state j, given that X_n is in state i (called the one step transition probability) is denoted by $p_{ij}(n,n+1)$, i.e.,

$$P_{ij}(n, n+1) = P[X_{n+1} = j / X_n = i] \quad \ldots\ldots\ldots (1.3.1)$$

The notation emphasizes that, in general, the transition probabilities are functions not only of the initial and final state, but also of the time of transition. When one step transition probabilities are independent of the time variable (i.e of the value of n), we say that the Markov Chain has stationary transition probabilities. Presently we will confine ourselves to such cases as the wast majority of Markov Chains that we shall encounter have stationary transition probabilities. In this case, $p_{ij}(n,n+1) = p_{ij}$ is independent of n and p_{ij} is the probability that the state value undergoes a transition from i to j in one trial. It is customary to arrange the numbers p_{ij} as a matrix.

$$P = \begin{bmatrix} p_{00} & p_{01} & p_{02} & \cdots \\ p_{10} & p_{11} & p_{12} & \cdots \\ p_{20} & p_{21} & p_{22} & \\ \vdots & \vdots & \vdots & \end{bmatrix} \qquad \cdots\cdot(1.3.2)$$

and refer to $P=(p_{ij})$ as the Markov matrix or transition probability matrix (t.p.m.) of the process.

The $(i+1)^{st}$ row of p is the probability distribution of the values of x_{n+1} under the condition $x_n=i$. If the number of states is finite p is a finite square matrix whose order is equal to the number of states. Clearly the quantities p_{ij}, satisfy the conditions

$$p_{ij} \geq 0, \quad i,j = 0, 1, 2, \ldots \qquad (1.3.3)$$

$$\sum_{j=0}^{\infty} p_{ij} = 1, \quad i = 0,1,2,\ldots \qquad (1.3.4)$$

The condition (1.3.4) shows that some transition occurs at each trial. (For convenience we say that a transition has occurred even of the state remains unchanged).

It can easily be shown, as indicated below, that the process is completely determined if (1.3.1) and the probability distribution of x_o are specified.

Let $P(x_o=i) = p_i$. Since any probability involving $x_{j1}.....,x_{jk}$, $j_1 < j_2 < ..... < j_k$, may be obtained, according to axiom of total probability by summing terms of the form

$$P[X_0 = i_0, X_1 = i_1, \ldots, X_n = i_n] \quad \ldots\ldots (1.3.5)$$

we will show as to how the quantities in (1.3.5) can be obtained.

By definition of conditional probabilities we obtain

$$P[X_0 = i_0, X_1 = i_1, \ldots, X_n = i_n]$$
$$= P[X_n = i_n / X_0 = i_0, X_1 = i_1, \ldots, X_{n-1} = i_{n-1}]$$
$$= P[X_0 = i_0, X_1 = i_1, \ldots, X_{n-1} = i_{n-1}] \ldots (1.3.6)$$

Now by the definition of Markov process

$$P[X_n = i_n / X_0 = i_0, X_1 = i_n, \ldots, X_{n-1}, i_{n-1}]$$
$$= P[X_n = i_n / X_{n-1} = i_{n-1}]$$
$$= P_{i_{n-1}, i_n} \quad \ldots\ldots (1.3.7)$$

substituting (1.3.7) in (1.3.6) we get

$$P[X_0 = i_0, X_1 = i_1, \ldots, X_n = i_n] = P_{i_{n-1}, i_n}$$
$$P[X_0 = i_0, X_1 = i_1, \ldots, X_{n-1} = i_{n-1}] \ldots (1.3.8)$$

By induction then it follows that

$$P[X_0 = i_0, X_1 = i_1, \ldots, X_n = i_n]$$
$$= P_{i_{n-1}, i_n} P_{i_{n-2}, i_{n-1}}, \ldots, P_{i_0 i_1}, P_i \quad (1.3.9)$$

CHAPMAN KOLMOGOROV EQUATION: A fundamental relation satisfied by the transition probability function

of a Markov chain $[x_n]$ is the Chapman-Kolmogorov equation, for any times $n > u > m \geq o$ and states j and k

$$P_{jk}(m,n) = \sum_{\text{states } i} P_{ji}(m,u)\, P_{ik}(u,n) \quad \ldots (1.3.10)$$

Summation in the equation is over all states of the Markov chain. To establish the equation we note the fact that

$$P[X_n = k / X_m = j] = \sum_{\text{states } i} P[X_n = k / X_u = i, X_m = j]\, P[X_u = i / X_m = j] \quad \ldots (1.3.11)$$

Applying Markov property we then find that:

$$P[X_n = k / X_u = i, X_m = j] = P[X_n = k / X_u = i]$$

and consequently we get

$$P_{jk}(m,n) = \sum_i P_{ii}(m,u)\, P_{ik}(u,n)$$

It can easily be shown that the continuous parameter Markov chain also satisfies the Chapman-Kolmogrov equation.

In term of multiplication of transition probability matrices, the Chapman-Kolmogrov equations for all times $n > u > m \geq o$ may be written as

$$P(m,n) = P(m,u).P(u,n)... \qquad (1.3.12)$$

where $P(m,n) = [p_{jk}(m,n)]$

and $p_{jk}(m,n) = P(X_n = k / X_m = j)$.

The probabilties $p_{jk}(m,n)$ refer to the probability of a transition from state j at time m to state k at time n, i.e., the number of steps involved is n-m. If $p_{jk}(m,n)$ depends only on the difference n-m, then the Markov chain is said to have stationary transition probability or to be homogeneous.

We then call

$$P_{jk}(n) = P[X_{n+t} = k / X_t = j] \quad \text{for all integers } t \geq o$$

The n step transition probability function of the homogeneous Markov chain $[X_n]$. In words, $p_{jk}(n)$ is the conditional probability that a homogeneous Markov chain. Now in state j will move after n steps to state k. The one step transition probabilities $p_{ij}(1)$, usually written as p_{ij}.

DETERMINING THE TRANSITION PROBABILITIES OF A MARKOV CHAIN

In case of descrete parameter Markav Chain, using the Chapman-Kolmogorov equation, we can derive various recurrence relations for the transition probability functions.

Let $[X_n]$ be a Markov Chain with transition probability matrix $[P(m,n)]$. From equation (1.3.12) it follows that

$$\begin{aligned} P(m,n) &= P(m,n-1) \cdot P(n-1,m) \\ &= P(m,n-2) \cdot P(n-2,n-1) \cdot P(n-1,n) \\ &= \cdots\cdots\cdots \\ &= \cdots\cdots\cdots \\ &= P(m,m+1) \cdot P(m+1,m+2) \cdots P(n-1,n) \cdots (13.13) \end{aligned}$$

Thus, to know P(m,n) for all $m \leq n$ it suffices to know the sequences of one step transition probability matrices

$$P(o,1),\ P(1,2),\ldots,P(n-1,n)\ldots.(1.3.14)$$

Next let us define the unconditional probability vector (for n = 0,1,2,...).

$$P^{(n)} = \begin{bmatrix} p_0^{(n)} \\ p_1^{(n)} \\ \vdots \\ p_j^{(n)} \\ \vdots \end{bmatrix}, \quad p_j^{(n)} = P(X_n = j) \cdots (1.3.15)$$

It is easily verified that

$$\underline{p}^{(n)} = P(0,u) \cdot \underline{p}^{(0)} \qquad \ldots\ldots\ldots\ldots (1.3.16)$$

In view of equations (1.3.16), (1.3.13) and (1.3.9) it follows that the probability law of a Markov Chain $[X_n]$ is completely determined once we know the transition probability matrices given in equation (1.3.14) and the unconditional probability vector $\underline{p}^{(o)}$ at time o.

In the case of homogeneous Markov chains X_n, let $P^{(n)} = [p_{jk}^{(n)}]$, $P = [p_{jk}]$ ..... (1.3.17) denote respectively the n-step of the one step transition probability matrices. From equation (1.3.13) and (1.3.16) it follows that

$$p^{(n)} = p^n \qquad (1.3.18)$$

$$\underline{p}^{(n)} = \underline{p}^{(n)} \cdot \underline{p}^{(o)} \qquad (1.3.19)$$

Consequently the probability law of a homogeneous Markov chain is completely determined once we know the one-step transition probability matrix $p=[p_{jk}]$ and the uncondition at probability vector $\underline{p}^{(o)} = (p_j^{(o)})$ at time o.

FINITE MARKOV CHAINS:

DEFINITION:

A Markov chain [Xn] is said to be a finite Markov Chain with K- states if the number of possible values of the random variable $[X_n]$ is finite and equal to K. The transition probabilities p_{jk} are then nonzero for only a finite number of values of j and k and the transition probability matrix P is then a kxk matrix.

TWO STATE MARKOV CHAIN:

Two states homogeneous Markov chains are

both simple and important. If the two states are denoted by 0 and 1, the transition probability matrix of a homogeneous two state Markov chain is of the form

$$P = \begin{bmatrix} p_{00} & p_{01} \\ p_{10} & p_{11} \end{bmatrix} \qquad \ldots\ldots\ldots (1.3.20)$$

The two step transition probability matrix is given by

$$P(2) = P^2 = \begin{bmatrix} p_{00}^2 + p_{01}p_{10} , p_{01}(p_{00}+p_{11}) \\ p_{10}(p_{00}+p_{11}) , p_{11}^2 + (p_{01}p_{11}) \end{bmatrix} \ldots (13.21)$$

In the case that $|p_{00}+p_{11}-1|<1$, it may be shown by mathematical induction that the n-step transition probability is given by

$$P(n) = \frac{1}{2-p_{00}-p_{11}} \begin{bmatrix} 1-p_{11} & 1-p_{00} \\ 1-p_{11} & 1-p_{00} \end{bmatrix} + \frac{(p_{00}+p_{11}-1)^n}{2-p_{00}-p_{11}} \begin{bmatrix} (1-p_{00}) & -(1-p_{00}) \\ -(1-p_{11}) & (1-p_{11}) \end{bmatrix} \ldots (13.22)$$

From equation (1.3.22) we obtain the simple asymptotic expression for the n-step transition probabilities

$$\lim_{n\to\infty} p_{00}(n) = \lim_{n\to\infty} p_{10}(n) = \frac{1-p_{11}}{2-p_{00}-p_{11}} \ldots (1.3.23)$$

$$\lim_{n\to\infty} p_{01}(n) = \lim_{n\to\infty} p_{11}(n) = \frac{1-p_{00}}{2-p_{00}-p_{11}} \ldots (1.3.24)$$

SECTION "B"

AN APPLICATION TO GENETICS

1.4 INTRODUCTION: In fact, the field of genetics provides an excellent ground for the application of stochastic models. The first fundamental application in this field is due to Fisher (1922,1930) who used the Galton-Watson model to study the survival of the progeny of a mutant gene. It was followed by a more general treatment by Kimura (1957) who introduced the method of the Kolmogorov backward equation to solve the problem. Since then this model has been modified remodified several times by a host of researchers cover a variety of problems of both theoretical and practical interest. A survey of the important applications of these models in the field of genetics is given by Schaffer (1970).

Genetics, the science of heredity, has been developed to explain the heredity of living things. Plant and animal breeders are always interested to develop new technology for genetic improvement. This improvement is possible by elimination of genes governing the undersirable characters and thus increasing the relative frequency of other desirable ones. In such programmes, assortative mating has the special merit over the random mating as the former results in an increase in Genotypic variance. The increased variance will give more opportunity for exercising selection in breeding programmes. To prepare a base for this work, we need the following few definitions.

DEFINITION 1.4.1. GENE:

The discrete particulate hereditary determiner located in the chromosome in linear order; the

"element" of Mendel and "factor' of early genetic terminology.

DEFINITION 1.4.2. GENOTYPE:

The genetic constitution or genetic make up of an organism is known as Genotype. When dominance is involved the Genotype can be determined by breeding the individuals.

DEFINITION 1.4.3 PHENOTYPE:

Phenotype is the appearance of an individual produced by the Genotype in cooperation with the environment. It is a contrasting term to Genotype.

DEFINITION 1.4.4. QUANTITATIVE GENETICS:

Quantitative Genetics is concerned with the inheritance of those characters that differ among individuals in degree rather than in kind, for example, the characters that exhibit differences in degree are growth rates in many species, milk production in dairy cattle, fleece weight in sheep etc.

DEFINITION 1.4.5 QUANTITATIVE INHERITANCE:

The quantitative characters are governed by a large number of minor genes, which are called as Polygenes, which are inherited in accordance with the Mendelian principles, each having a small, similar and cumulative effects, and whose effects are highly susceptible to environmental modifications. Such inheritance is also called polygenic inheritance.

DEFINITION 1.4.6. RELATIVE FITNESS:

Relative fitness of a genotype is the relative contribution of that genotype to the genetic composition of the next generation.

DEFINITION 1.4.7 MIGRATION:

Migration occurs when a large influx of individuals moves into another population and interbreeds with the later.

DEFINITION 1.4.8. MUTATION:

The sudden heritable changes in genes other than those due to Mendelian Segregation and recombination constitute mutation.

DEFINITION 1.4.9 SELECTION:

Selection is the process whereby some individuals get more chances for reproduction. It occurs due to difference in fitness of the genotype. It is one of the forces that changes gene frequencies in the population and a fundamental process of evolutionary change.

DEFINITION 1.4.10 DOMINANCE:

Complete suppresion of the expression of one allele by another at the same locus in the chromosome is termed as dominance.

DEFINITION 1.4.11 RECESSIVE:

A term used by Mendel to describe characters which recede completely in the F_1. Action of the recessive allele is suppressed by the dominant one.

DEFINITION 1.4.12 RANDOM MATING:

A mating in which the individuals choose their mates independent of their genetic constitution.

DEFINITION 1.4.13 GENOTYPIC ASSORTATIVE MATING:

A mating is said to be assortative genotypically if the mated pairs are more similar genotypically than if they were chosen at random from the population.

DEFINITION 1.4.14 PHENOTYPIC ASSORTATIVE MATING:

A mating is said to be assortative phenotypically if the mated pairs are more similar for some phenotypic trait than if they were chosen at random from the population.

DEFINITION 1.4.15 GENOTYPIC VARIANCE:

The variance of any trait which is determined solely by Genetic differences among the individuals in the population is called Genotypic variance. It is the total hereditary constribution to the variance.

CHAPTER-II

MODELS FOR PUPULATION GROWTH

2.1. INTRODUCTION:

Biology studies complex situations and therefore needs skilful methods of abstraction. Stochastic models, being both vigorous in their specification and flexible in their manipulation, are the most suitable tools for studying such situations.

Time, life and risks are three basic elements of stochastic processes in biostatistics. Risks of death, risks of illness, risks of birth and other risks act continuously on man with varying degrees of intensity.

It is the characterization of Lewontin (1963) concerning stochastic models of biological populations, certainly real populations of plants and animals are not infinitely large and the number of effective parents of each generation may be much smaller than the total population, as in social units of bees. Moreover, the environment is not usually constant but undergoes a certain amount of random fluctuation. A more realistic group of models is then one in which the role of chance in determining which individual will have what sort of offspring is taken into account. There are, of course, the stochastic models.

Stochastic models generally deals with the description and prediction of events taking place within populations including events leading to the constitution of the population itself.

We shall consider those populations which represent sets of objects with a determined and complex character i.e. biological objects and describe some of the models for population growth.

2.2. SOME STOCHASTIC MODELS OF POPULATION GROWTH

A stochastic process x(t) is a random variable in which the parameter "t" is often interpreted as time, is real, lead may be either descrete or continuous. The random variable x(t) may be real valued or complex valued. In the study of the population growth we shall have a continuous time parameter "t" but the random variable x(t) will have a discrete set of possible values.

Let [X(t), $t \in T$] be a stochastic process, with "T" as the parameter space and "S" as the state space. Define the conditional distribution function of X(t) as

$$P(x_0, x_1; t_0, t_1) = P[X(t_1) \leq x_1 / X(t_0) = x_0], \quad t_0, t_1 \in T,$$

$$\text{and} \quad x_0, x_1 \in S, \; t_0 < t$$

consider an arbitrary finite (or countably infinite) set of points

$$(t_0, t_1, \ldots\ldots, t_n, t), \; t_0 < t_1 < t_2 < \cdots < t_n < t$$

and $t, t_r \in T \quad (r = 0, 1, \ldots, n)$.

The process X (t) is then a Markov process if we can write $P[X(t) \leq x / X(t_n) = x_n, X(t_{n-1}) = x_{n-1}, \ldots, X(t_0) = x_0]$

$$= P[X(t) \leq X / X(t_n) = X_n]$$
$$= P[X_n, X; t_n, t]$$

To begin with suppose that the population being considered is, at any instant of t, represented by a discrete random variable X(t) with

$$P[X(t) = n] = P_n(t), \quad n = 0, 1, 2, \ldots \qquad 2.2.1$$

The formulation of a practical problem in terms of stochastic processes involves to simplify some assumptions. Such formulation often produces enlightening and useful answer to the problem. At certain instants of time there will be discrete changes in the population size, due to for example to the loss or death of an individual, as to the appearance or birth of new individuals. To fix ideas, a few basic simple applicable stochastic processes are introduced in this section, which are commonly used as models of population growth.

POI SSON PROCESS: Let X(t) be the numberof events occurring in the time interval (0,1). Then X(t) is called a renewal process if the time intervals between two consecutive occurrence of events are independent and identically distributed random variables. Furthermore, if the time interval between two consecutive occurrence of events in renewal process are independent , i dentically distributed random variable and with probability density function.

$$f(X) = \lambda e^{-\lambda x}, \quad X > 0$$

the renewal process "t" Poisson process. Its form is given by

$$P_n(t) = PX(t) = n = \frac{e^{-\lambda t}(\lambda t)^n}{n}, \quad n = 0, 1, \ldots \quad (2.2.2)$$

Basic assumptions for the Poisson process are : for all $t > 0$

i) events occurring in non-overlapping intervals of time are independent of each other,

ii) for a sufficiently small Δt there is a constant λ (which is a parameter depending on the intensity of the arrivals), such that the probabilities of occurrence of the events in the interval $[t, t+\Delta t]$ are given as follows:

a) $P_{ii}(t, t+\Delta t) = 1 - \lambda \Delta t + o(\Delta t) \quad \ldots (2.2.3)$

b) $P_{i,i+1}(t, t+\Delta t) = \lambda(t) + o(\Delta t) \quad \ldots (2.2.4)$

c) $\sum_{j=i+2}^{\infty} P_{ij}(t, t+\Delta t) = o(\Delta t) \quad \ldots (2.2.5)$

d) $P_{ij}(t, t+\Delta t) = 0 \quad \ldots (2.2.6)$

where $o(\Delta t)$ contains all terms that tend to zero, much faster than Δt, ie $\lim_{\Delta t \to 0} \frac{o(\Delta t)}{\Delta t} = 0 \quad \ldots (2.2.7)$

Consider two adjacent intervals $(0,t]$ and $(t, t+\Delta t]$. The occurrence of exactly n events during the intervals $(0, t+\Delta t)$ can be realised in three mutually exclusive ways, (i) all events will occur in $(0,t]$ and none in $(t, t+\Delta t]$ with probability $P_n(t)[1 - \lambda \Delta t + o(\Delta t)]$ (ii) exactly $n-1$ events will occur in $(0,t]$ and one event in $(t, t+\Delta t]$

with probability $P_{n-1}(t)\left[\lambda\Delta t + o(\Delta t)\right]$, and (iii) exactly $(n-k)$ events will occur in $(0, t]$ and k events in $(t, t+\Delta t]$, where $2 \leq j \leq k$, with the probability $o(\Delta t)$.

Hence combining all quantities of order $o(\Delta t)$ we have for $n > 0$

$$P_n(t+\Delta t) = P_{on}(0, t+\Delta t) \text{ for } n > 0$$
$$= P_n(t)\left[1-\lambda\Delta t\right] + P_{n-1}(t)\lambda\Delta t + o(\Delta t) \quad \ldots (2.2.8)$$

and for $n = 0$.

$$P_0(t+\Delta t) = P_0(t)\left[1-\lambda\Delta t\right] + o(\Delta t) \quad \ldots (2.2.9)$$

transposing $P_0(t)$ in (2.2.9) and $P_n(t)$ in (2.2.8) and dividing both the equations by Δt and taking limit in both the equations as $\Delta t \to 0$, we get

$$\lim_{\Delta t \to 0} \frac{P_0(t+\Delta t) - P_0(t)}{\Delta t} = -\lambda P_0(t) + \lim_{\Delta t \to 0} \frac{o(\Delta t)}{\Delta t}$$

$$\lim_{\Delta t \to 0} \frac{P_n(t+\Delta t) - P_n(t)}{\Delta t} = -\lambda P_n(t) + \lambda P_{n-1}(t) + \lim_{\Delta t \to 0} \frac{o(\Delta t)}{\Delta t}$$

or $P_0'(t) = -\lambda P_0(t) \quad n = 0 \quad \ldots (2.2.10)$

$$P_n'(t) = -\lambda P_n(t) + \lambda P_{n-1}(t), \quad n > 0 \quad \ldots (2.2.11)$$

with initial conditions

$P_0(0) = 1$, $P_n(0) = 0 \quad n \geq 1$

Integrating (2.2.10) and using the initial condition $P_0(0) = 1$ Yields

$$P_0(t) = e^{-\lambda t} \quad \ldots (2.2.12)$$

putting $n = 1$ in (2.2.11) and multiplying both sides by e^{it}, we get

$$e^{\lambda t} P_1(t) + \lambda e^{\lambda t} P_1(t) = \frac{d}{dt}\left[e^{\lambda t} P_1(t)\right] = \lambda \quad \ldots (2.2.13)$$

Integrating and using $P_1(0) = 0$, we have

$$P_1(t) = \lambda t e^{-\lambda t}$$

Repeated applications of the same procedure yields the general formula

$$P_n(t) = \frac{e^{-\lambda t}(\lambda t)^n}{n!} \quad , \quad n = 0, 1, 2, \ldots\ldots$$

PURE BIRTH PROCESS: There are several problems of population growth for which a certain class of simple Markov Process Called "birth Process" has been found to be useful as mathematical model. In a birth process model, assumptions are made of the nature of increased population such that the resulting process is a reasonable model if we are interested only in the number of people infected. In the poisson process the parameter λ remains constant irrespective of the population size. Pure birth process is obtained by making the parameter λ depend on n. Let us suppose that X (t) = n, then the probabilities of occurrence of events, are given as (i) The probability that a new event will occur during $(t, t + \Delta t]$ is $\lambda_n \Delta t + 0(\Delta t)$ where λ_n is some function of n.

(ii) The probability that more than one events will occur in $(t, t + \Delta t)$ is $0(\Delta t)$.

(iii) The probability that the event does not occur in $(t, t + \Delta t]$ is $1 - \lambda_n \Delta t + 0(\Delta t)$.

Let $P_n(t)$ be the probability that "events occur in time $(0, t]$. Using Chapman - Kolomogorov equations for transitions in the intervals of time $(0, t]$ and $(t, t + \Delta t]$, we can write

$$P_0(t + \Delta t] = P_0(t)[1 - \lambda_0 \Delta t] + o(\Delta t)$$

$$P_n(t + \Delta t] = P_n(t)[1 - \lambda_n \Delta t] + P_{n-1} \lambda_{n-1} \Delta t + 0(\Delta t)$$

$$n > 0 \quad \ldots\ldots (2.2.14)$$

which gives

$$P_0'(t) = -\lambda_0 P_0(t)$$
$$P_n'(t) = -\lambda_n P_n(t) + \lambda_{n-1} P_{n-1}(t), \quad n > 0 \qquad \ldots\ldots (2.2.15)$$
With $P_0(0) = 1$ and $P_n(0) = 0$ for $n > 0$.

These differential equations may be solved to give

$$P_n(t) = \sum_{v=0}^{i} A_n(v) e^{-\lambda_v t} \quad n \geq 0 \qquad \ldots\ldots (2.2.16)$$

Where

$$A_n(v) = \frac{\lambda_0 \lambda_1 \ldots \lambda_n}{(\lambda_0 - \lambda_v)(\lambda_1 - \lambda_v)\ldots(\lambda_{v-1} - \lambda_v)(\lambda_{v+1} - \lambda_v)\ldots(\lambda_n - \lambda_v)} \qquad \ldots\ldots (2.2.17)$$

It can also be shown that $P_n(t)$ is proper distribution

$$\left[\sum_{n=0}^{\infty} P_n(t) = 1\right] \text{ if and only if } \sum_{v=0}^{\infty} \lambda_v^{-1} = \infty$$

<u>THE YULE PROCESS</u>: It is a special case of pure birth process. By representing λ_n by $n\lambda$ we can write the equation (2.2.15) as

$$P_n'(t) = n\lambda P_n(t) + (n-1)\lambda P_{n-1}(t) \quad n > 0 \qquad \ldots\ldots (2.2.18)$$

Assuming that $P_1(0) = 1$ and $P_i(0) = 0$ for $i > 1$, the solution can be obtained.

$$P_n(t) = \sum_{v=1}^{\infty} A_n(v)_e - v\lambda t \qquad \ldots\ldots (2.2.19)$$

where

$$A_n(v) = \frac{1.2.\ldots.n-1}{(1-v)(2-v)\ldots(v-1-u)(v-1+u)\ldots(n-1)} \qquad \ldots\ldots (2.2.20)$$

Or

$$A_n(v) = \frac{\lfloor n-1 \; (-1)^{v-1}}{\lfloor v-1 \; \lfloor n-v} = (-1)^{v-1} \binom{n-u}{v-1}$$

Thus we find $P_n(t) = e^{-\lambda t} \sum_{v=1}^{n} \binom{n-1}{v-1} (-e^{-\lambda t})^{v-1}$

or $P_n(t) = e^{-\lambda t}(1 - e^{-\lambda t})^{v-1}$ ..... (2.2.22)

Note that $P_n(t)$ is geometric with $P = e^{-\lambda t}$

and $q = 1-e^{-\lambda t}$

THE POLYA PROCESS: The Polya process is a non-homogeneous birth process in which birth occurs in the time interval $(t,t+\Delta t)$ was assumed to be $\lambda_n \Delta t + o(\Delta t)$. Suppose that λn is a function of both n and t such that

$$\lambda_n(t) = \frac{\lambda + \lambda an}{1 + \lambda an} \qquad \text{.....} (2.2.23)$$

Where λ and a are nonhomogeneous constants with $X(0) = n_o$, the differential equations for the probability distribution are

$$P'_{n_o}(t) = \frac{r + \lambda a n_o}{1 + \lambda at} P_{n_o}(t)$$

$$P'_n(t) = \frac{\lambda + \lambda an}{1 + \lambda at} P_n(t) + \frac{\lambda + \lambda a(n-1)}{1 + \lambda at} P_{n-1}(t), \qquad n > n_o \quad \text{.....} (2.2.24)$$

We shall use P.g.f. to solve the above equation

$$G_x(s,t) = \sum_{n=n_o}^{\infty} P_n(t)\ s^k \qquad \text{.....} (2.2.25)$$

with

$$G_x(S,0) = S^{n_o} \qquad \text{.....} (2.2.26)$$

We take the derivative of $G_x(s,t)$ with respect to t and use the system of equations (2.2.24)) to obtain the partial differential equation.

$$(1+\lambda at)\frac{\partial}{\partial t} G_x(S,t) + \lambda as(1-S)\frac{\partial}{\partial S} G_x(S,t) = -\lambda(1-S) G_x(S,t) \qquad \text{.....} (2.2.27)$$

The auxiliary equations are

$$\frac{dt}{1+\lambda} = \frac{ds}{\lambda as(1-s)} = \frac{d\,G_X(s,t)}{-\lambda(1-s)G_X(s,t)} \quad \cdots\cdots (2.2.28)$$

The first equation may be written as

$$\frac{dt}{1+\lambda at} = \frac{1}{\lambda a}\left[\frac{1}{s} + \frac{1}{1-s}\right] ds \quad \cdots\cdots\cdots (2.2.29)$$

or

$$d\log(1+\lambda at) = d\log\frac{s}{1-s}$$

with the solution

$$\frac{1-s}{s}(1+\lambda at) = \text{constant} \quad \cdots\cdots (2.2.30)$$

From the second auxiliary equation, we have

$$\frac{1}{a} d\log s = -\,d\log G_X(s,t) \quad \cdots\cdots (2.2.31)$$

so that

$$G_X(s,t)\, s^{1/a} = \text{constant} \quad \cdots\cdots\cdots (2.2.32)$$

Hence the general solution of (2.2.27) is

$$G_X(s,t) = s^{-1/a}\,\phi\left[\left(\frac{1-s}{s}\right)(1+\lambda at)\right] \quad \cdots\cdots (2.2.33)$$

where ϕ is an arbitrary differential function putting $t = o$ in (2.2.33) and use (2.2.26) to write

$$s^{-1/a}\,\phi\left[\frac{1-s}{s}\right] = s^{n_o} \quad \cdots\cdots\cdots (2.2.34)$$

equation (2.2.34) hold true for all s such that $|s|<1$,

hence for any θ, $\left|\frac{1}{1+\theta}\right| < 1$,

$$\phi\left[\frac{1-s}{s}\right] = s^{n_o+\frac{1}{a}}$$

$$\phi\left[\frac{1-\frac{1}{1+\theta}}{1/1+\theta}\right] = \left[\frac{1}{1+\theta}\right]^{n_o+\frac{1}{a}} \quad \cdots\cdots\cdots (2.2.35)$$

$$\phi(\theta) = (1+\theta)^{-(n_o+\frac{1}{a})}$$

$$\text{let } \theta = \frac{1-s}{s}(1+\lambda at).$$

Therefore from (2.2.34), we have the required solution for the p.g.f.

$$G_X(s,t) = s^{-1/a}\left[1 + \frac{1-s}{s}(1+\lambda at)\right]^{-(n_o+\frac{1}{a})}$$

$$= s^{n_o}\left[\frac{1/(1+\lambda at)}{1-s[\lambda at/(1+\lambda at)]}\right]^{K_o+\frac{1}{a}} \quad \cdots\cdots\cdots (2.2.36)$$

Hence X(t), is except n_o, a negative binomial random variable with parameter

If we write (2.2.36) as

$$G_X(s,t) = \left\{\frac{s}{1+\lambda at-\lambda ats}\right\}^{k_0}\left\{1+\lambda at-\lambda a/s\right\}^{-1/a} \quad \ldots\ldots (2.2.37)$$

it is clear that

$$\lim_{a\to 0} G_X(s,t) = s^{k_0} e^{-(1-s)\lambda t} \quad \ldots\ldots (2.2.38)$$

Thus as $a \to o$, the Polya process tends to poisson process.

PURE DEATH PROCESS: The physical phenomenon in which there is no increase in the population size, when once the process starts, is called "Pure death process". For instance consider a group of customers who enter the side walk from a single store, they leave the side walk after some time or consider a retailer, who starts with a certain inventory. His inventory gets depleted in the course of time and needs restocking. The mathematical model suited for such situations is the pure death process. Let $X(t)=n$, the death rate μ_n, defined as follows:

In the interval $(t,t+\Delta t)$ the probability that one death occurs is $\mu \Delta t + O(\Delta t)$, the probability that no death occurs is $1 - \mu_n \Delta t + o(\Delta t)$ and the probability that more than one death occurs in $(t, t + \Delta t)$ is $o(\Delta t)$. Also assume that occurrence of death in $(t, t + \Delta t)$ is independent of time since the last death. For such a process we can derive the last death. For such a process we can derive the difference differential equation as follows:

Considering the transitions occurring in the intervals $(0, t)$ and $(t, t + \Delta t)$ we have

$$P_i(t+\Delta t) = P_i(t)[1-\mu_n \Delta t + o(\Delta t)], \quad i>0$$

$$P_n(t+\Delta t) = P_n(t)[1-\mu_n \Delta t + o(\Delta t)] + \mu_{n+1}\Delta t\, P_{n+1}(t) + O(\Delta t), \quad n>i \quad \ldots\ldots (2.2.39)$$

giving

$$P_i'(t) = -\mu_i P_i(1) \quad \ldots\ldots (2.2.40)$$

$$P_n'(t) = -\mu_n P_n(t) + \mu_{n+1} P_{n+1}(t)$$

In the special case $\mu_n = n\mu$ the solution to these equations takes from

$$P_n(t) = \binom{i}{n} e^{-n\mu t}(1 - e^{-\mu t})^{i-n} \quad n \geq i \qquad \ldots\ldots\ldots(2.2.4i)$$

which is clearly a binomial distribution with $P = e^{-\mu t}$ and $q = 1 - e^{-\mu t}$

BIRTH AND DEATH PROCESS:: A process which allow increase and decrease both in the population size with Markov property is considered to be a birth and death process.

Consider a group of customers who enter the sidewalk from several stores, a birth death process cannot be a realistic mathematical model. Because of the possible increase and decrease in the population size, a process that would allow for such variations must be considered, one such model with Markov properties is the so called "birth and death process". This has been proved to be realistic and used in many problems related to the spread of epidemics, composition in queueing systems, such as telephone trunking, traffic and maintenance problems etc.

Let x(t) denote the population size at time t for $0 \leq t \leq \infty$, with the critical size x(t)=n may be classified as follows:

i) chance of one birth during interval $[t, t + \Delta t]$ is $\lambda_n \Delta t + o(\Delta t)$

ii) chance of one death in interval $[t, t + \Delta t]$ is $\mu_n \Delta t + O(\Delta t)$.

iii) chance of more than change is $O(\Delta t)$, and

iv) chance of no change in $[t, t + \Delta t]$ is $1 - \lambda_n \Delta t + O(\Delta t)$.

Consequently the probabilities $P_n(t + \Delta t)$ at time $(t + \Delta t)$ may be expressed in the form of differential equations.

$$P_n(t+\Delta t) = P_n(t)[1 - \lambda_n(\Delta t) - \mu_n \Delta t] + P_{n-1}(t)\lambda_{n-1}\Delta t + P_{n+1}(t)\mu_{n+1}\Delta t + o(\Delta t) \quad \ldots\ldots(2.2.43)$$

which on re-arranging and letting $t \longrightarrow 0$ gives the difference differential equation.

$$P_n'(t) = -(\lambda_n + \mu_n)P_n(t) + \lambda_{n-1}P_{n-1}(t) \quad \ldots\ldots(2.2.44)$$

with the initial condition

$$P_n(0) = \begin{cases} 1 & \text{if } n = i \\ 0 & \text{if } n \neq i \end{cases}$$

The solution of this differential equation take a complicated form. Suppose that $n=n\lambda$ and $\mu_n = n\mu$ (i.e. when both the increase and decrease in the population is linearly dependent on the population size) for i=o, we get

$$P_0(t) = \xi_t = \mu \frac{1 - e^{-(\lambda-\mu)t}}{\lambda - \mu e^{-(\lambda-\mu)t}} \quad \ldots\ldots(2.2.45)$$

$$P_n(t) = (1 - \xi_t)(1 - \eta_t)\eta_t^{n-1} \quad \ldots\ldots(2.2.46)$$

where

$$\eta_t = (\lambda/\mu)\xi_t = \lambda\left[1 - e^{(\lambda-\mu)t}\right] \Big/ \left[\lambda - \mu e^{-(\lambda-\mu)t}\right]$$

Clearly, $P_n(t)$ has a geometric distribution with a modified initial term.

When the increase in the population is linearly dependent on the size of the population, it is pertinent to investigate the population would ever become extinct. The behaviour of

the probability $P_0(t)$ gives due to the probability, for $\lim_{t\to\infty} P_0(t)$ = Probability of ultimate extinction of the Population.

Let $\rho = \lambda/\mu$, then from (2.2.45), we have

$$\lim_{t\to\infty} t = \lim_{t\to\infty} \frac{\mu\left[1-e^{-(\lambda-\mu)t}\right]}{\lambda - e^{-(\lambda-\mu)t}} = \frac{\mu}{\lambda} \quad \rho > 1 \qquad \ldots(2.2.47)$$

and $\lim_{t\to\infty} \rho t = \lim_{n\to\infty} \frac{e^{(\mu-\lambda)t}\left[\mu e^{-(\mu-\lambda)t} - \mu\right]}{e^{(\mu-\lambda)t}/\lambda e^{-(\mu-\lambda)t} - \mu} = 1 \; ; \rho \le 1$

Thus we have $\ldots(2.2.48)$

$$\lim_{t\to\infty} P_0(t) = \begin{cases} 1 & \text{if } \rho \le 1 \\ \rho - 1 & \text{if } \rho > 1 \end{cases} \qquad \ldots\ldots\ldots\ldots\ldots (2.2.49)$$

which is in accordance with the initiative result that when the death rate is larger than the birth rate ultimate extinction is certain

2.3 : In the last decade several authors have examined various aspects of Growth models. In the following sub sections we present a brief account of some of the more recent contributions.

AGE DEPENDENT MODELS OF POPULATION GROWTH

The simplest mathematical models of population growth consider whole populations, with no particular distinction between individuals. Time can either be measured in discrete units, leading to a difference equation $x_{n+1} = \lambda x_n'$, or continuously, leading to a differential equation $X' = \rho x$. The growth factor λ is 1 + b — d, if we suppose that there are b births per individual per year and d deaths. The instantaneous per capital annual growth rate ρ corresponds to $\lambda - 1$. When these vital rates depend on population size, one is led to the logistic growth equations.

$$X_{n+1}' = [\, 1 + (\lambda - 1)\, X_n/k \,]^{-1} \lambda X_n \qquad (2.3.1)$$

and

$$X' = \rho(1 - x/k)x. \qquad (2.3.2)$$

Bernardelli (1941), Lewis (1942), and Leslie (1945) replaced the scalar difference equation with a vector-matrix equation to take account of the variation of birth and death rates with age. Thus, suppose that after n years a population contains x_{1n} individuals whose ages lie in the interval $(0,r_1)$ years, X_{2n} whose ages lie in the interval $[r_1, r_1+r_2]$ years,...X_{4n} aged $r_1 +....+ r_3$ and older (we use four age classes to simplify notation). Birth and death rates are assumed uniform in each class, b_1, d_1,...b_4, d_4. Thus if

$$L=\begin{bmatrix} (1-1/r_1)(1-d_1)+b_1 & b_2 & b_3 & b_4 \\ (1-d_1)/r_1 & (1-1/r_2)(1-d_2) & 0 & 0 \\ 0 & (1-d_2)/r_2 & (1-1/r_3)(1-d_3) & 0 \\ 0 & 0 & (1-d_3)/r_3 & 1-d_4 \end{bmatrix}$$

the population vector $X_n = (X_{1n},...,X_{4n})$ satisfies the unrestricted growth difference equation $X_{n+1}=LX_n$. This equation says, for example, that

$$X_{1(n+1)} = (1-1/r_1)(1-d_1)X_{1n} + b_1X_{1n} + \cdots + b_4X_{4n}$$

The sum $b_1x_{1n}+....+b_4x_{4n}$ represents births to this year's population, and the remaining term counts the members of the first age group who neither die nor "graduate" to the next.

This difference equation is satisfied by $X_n=L^nX_o$, and the structure of L determines how the solution behaves as time goes on (n increases). To avoid detours outside our area of interest, let us assume that b_4 is positive and that the d's are all less than 1 (no age class has a 100% death rate). Then L is an irreducible matrix with nonnegative entries and hence has a positive simple characteristic value λ which is not exceeded by the magnitude of any other

characteristic value. Corresponding to λ are positive characteristic vectors p and q of L and L* (L transpose). Sykes (1969) discusses the possibility that L has another characteristic value of magnitude λ , but we will suppose that it doesn't. Then for large n,

$$X_n \simeq \lambda^n \langle q, x_0 \rangle \langle q, p \rangle^{-1} p \quad \ldots (2.3.3)$$

Approximation (2.3.3) shows that p gives the proportions of the "ultimate" population that lie in the different age classes. Observe that if u= (1,...,1), then $L^*u = (1+b_1-d_1, \ldots 1+b_4-d_4)$ = u+b-d. Also, $(p, L^*u)=(Lp,u)= \lambda (p,u)$, and so $\lambda - 1 = (p, b-d)/(p,u)$. Thus $\lambda - 1$ represents the excess of birthrates over deathrates, as weighted by the ultimate population. For true growth, we will require that λ exceed 1, equivalently, det $(I-L) < 0$.

A vector version of (2.3.1) was introduced by Leslie (1948),

$$X_{n+1} = \left[1 + \langle (L-1)X_n, W \rangle / k\right]^{-1} L X_n \quad \ldots (2.3.4)$$

Here w is a "weight vector," and k is the "carrying capacity of the environment." We will discuss these quantities (and also explain why our version of Leslie's logistic equation seems to differ from his) when we get to the vector logistic differential equation. Our contribution to the theory of (2.3.4) is the observation that it has a closed-form solution,

$$X_n = \left[1 + \langle (L^n - 1)X_0, W \rangle / k\right]^{-1} L^n X_0$$

From this expression and (2.3.3), we see that

$$\lim_{n \to \infty} X_n = k \langle p, W \rangle^{-1} p .$$

To go from our difference equations to differential equations, we start by "compounding" the growing population more often than once a year, say, m times. Then the population vectors X_{n+1} and X_n are 1/m years apart, and the growth and death rates for the period will be b_i/m and d_i/m. In place of L we have the growth matrix $L_m = 1 + m^{-1}U_m$, where

$$U_m = \begin{bmatrix} b_1 - 1/r_1 - d_1 + d_1/mr_1 & b_2 & b_3 & b_4 \\ 1/r_1 - d_1/mr_1 & -1/r_2 - d_2 + d_2/mr_2 & 0 & 0 \\ 0 & 1/r_2 - d_2/mr_2 & -1/r_3 - d_3 + d_3/mr_3 & 0 \\ 0 & 0 & 1/r_3 - d_3/mr_3 & -d_4 \end{bmatrix}$$

When n periods of time have elapsed, the population vector is $X_n = L^n{}_m X_o$. After t years, n = mt, so

$$X_{mt} = L_m^{mt} X_o = \left[I + \frac{1}{m} U_m\right]^{mt} X_o = \left[I + \frac{t}{mt} U_m\right]^{mt} X_o .$$

Now let $m \uparrow \infty$ and this vector becomes $e^{Rt} x_o$, where $R = U_\infty$.

The vector $X = e^{Rt} X_o$ satisfies the unrestricted vector growth differential equation.

$$X' = Rx. \qquad \text{..... } (2.3.5)$$

The coefficient matrix R has a simple real characteristic value ρ which exceeds the real part of any other characteristic value. If corresponding positive characteristic λ vectors of R and R* are again denoted by p and q, it will be true that, for large t,

$$e^{Rt}x_0 \simeq e^{\rho t}\langle q, x_0\rangle\langle q, p\rangle^{-1}p \quad \ldots\ldots (2.3.6)$$

All entries of the matrix e^{Rt} are positive. To assure growth, we suppose that $\rho > 0$, which is equivalent to det $(-R) < 0$.

We will take our vector logistic differential equation to be

$$x' = [R - k^{-1}\langle Rx, w\rangle I]x \quad \ldots\ldots (2.3.7)$$

For example, suppose that we are observing a school of fish and that the weight vector's components $w_1, \ldots, w_4$ are the average weights of the fish in the various age classes. Since x gives the numbers of fish, (x,w) is their total weight. These fish inhabit waters that will support a total of k pounds. If we were interested in total numbers of fish (rather than weight), we would replace w with u. Choosing w and k is a biological problem; mathematically, it would be simpler to write v in place of $k^{-1}R^*w$. Thus Leslie's version of (2.3.4) is obtained from ours by letting $w = (\lambda - 1)(L-1)^{-1*}u$.

Equation (2.3.7) seems to be a direct generalization of the scalar logistic equation (2.3.2). There the growth factor $\rho - \rho x/k$ is obtained from the natural growth rate ρ by subtracting the rate of growth of the fraction of the carrying capacity that is being utilized. In the vector case, that quantity is (Rx, w)/k.

Although (2.3.7) is a nonlinear differential equation, we can write down its solution explicitly,

$$X = [1 + \langle (e^{Rt} - I)X_0, W/k]^{-1} e^{Rt} X_0 \quad \cdots (2.3.8)$$

From this equation and (2.3.5), we see that

$$\lim_{t \uparrow \infty} X = K \langle P, W \rangle^{-1} P \quad \cdots (2.3.9)$$

Thus the constant solution $x = k(p,w)^{-1}p$ is an attractor and represents a stable population weighing k pounds. To avoid a zero denominator in (2.3.3), we may have to require that x_0 point "closely enough" in the critical direction P or that (x_0, w) not be "too large."

Observe from (2.3.3) that

$$\langle X, W \rangle - K = [1 + \langle (e^{Rt} - I)X_0, W \rangle / K]^{-1} (\langle X_0, W \rangle - K).$$

Therefore, the sign of $(x,w) - k$ does not change; there are always fewer than k pounds of fish, exactly k pounds, or more than k pounds, depending on the starting state.

Even when R and w depend on t, (2.3.7) can be solved explicitly in terms of a solution of (2.3.5). For example, if w is constant and U is the matrix solution of (2.3.5) such that $U(0) = I$, then (2.3.7) is satisfied by

$$X = [1 + \langle (U(t) - I)X_0, W \rangle / K]^{-1} U(t) X_0 \quad \cdots (2.3.10)$$

Let us suppose that the vital rates b and d are p-periodic functions of t. To be sure that we have all the consequences of irreducibility, we will assume that R(t) is bounded below by

a constant matrix of the type we have just been disussing. Floquet theory says that (2.3.5) has a fundamental solution $U(t)=P(t)e^{Ct}$, where $P(t)$ has period $2p$ and C has real entries. Because e^{Ct} is positive (and indeed has unbounded entries) for sufficiently large t, we can infer that C shares some of the properties of our earlier constant R. In particular, there are positive vectors a and b and a positive scalar γ such that $e^{Ct}x_0 \simeq e^{\gamma t}(b, x_0)(b,a)^{-1} a$ for large t, and hence $U(t)x_0 \simeq e^{\gamma t}(b,x_0)\,(b,a)^{-1} P(t)a$.
When this result is substituted in (2.3.10), we find that

$$x \simeq k(P(t)a,w)^{-1} P(t)a.$$

Thus as time goes on, the total mass (x,w) of fish tends to the carrying capacity k, but the populations of the different age classes ebb and flow periodically.

Finally, we remark that still more complicated nonlinear equations can be solved more or less explicitly in terms of solutions of (2.3.5). Suppose, for example, that we harvest h fish per year, taking the same fraction of each age group. Then (2.3.7) becomes (with constant R, for simplicity)

$$x' = [R-(\langle Rx,w\rangle/k+h/\langle x,u\rangle)/I]x \quad \text{---(2.3.11)}$$

(If h pounds were harvested, we would replace u with w). It is simply a matter of substitution to see that this equation is satisfied by $x = \phi(t)e^{Rt}x_0$, where $y = \phi(t)$ satisfies the scalar initial value problem

$$y' = -\langle Re^{Rt}x_0, w\rangle y^2/k - h/\langle e^{Rt}x_0, u\rangle \text{ and}$$
$$Y=1 \text{ when } t=0 \quad \text{.....(2.3.12)}$$

By substituting $x = \beta p$, we find that (2.3.11) has equilibrium solutions $x = \beta_1 p$ and $x = \beta_2 p$ where β_1 and β_2 are solutions of a certain quadratic equation. As in the scalar case, the solution corresponding to the larger β (say β_1) is an attractor. That is, if x_0 is sufficiently close to $\beta_1 p$, then $\lim_{t \to \infty} x = \beta_1 p$. This statement can be established by observing that, in view of (2.3.6), it is equivalent to $\lim_{t \to \infty} \phi(t) e^{\rho t} = \beta_1 \langle q, p \rangle / \langle q, x_0 \rangle$.

We verify this latter limit by analyzing (2.3.12). The condition that β_1 and β_2 be distinct real numbers is that $h < \rho k \langle p, u \rangle / 4 \langle p, v \rangle$. Observe from (2.3.9) that the right hand side of this inequality is $\rho/4$ times the total number of fish that would be present at equilibrium if there were no harvest.

2.4. EFFECT OF SEASONALITY ON DISCRETE MODELS OF POPULATION GROWTH

The effects of seasonality on the dynamics of a bivoltine population with discrete, non-overlapping generations are examined here.

Non-linear difference equations have been used for many years in the study of the growth of populations. They have appeared as models for populations with discrete, nonoverlapping generations (e.g., Nicholson & Bailey, 1935) and as approximate growth equations for species with continuous reproduction and mortality (Ricker, 1954). More recently (May, 1974, 1975, 1976; May

and Oster, 1976; Guckenheimer et al., 1977; Rodgers, 1981), scientists have focused attention on the wide range of dynamics inherent in these equations. It is now known that even first order, one dimensional, difference equations may exhibit "chaotic" solutions. Chaotic trajectories are asymptotically aperiodic. They are often visually indistinguishable from a sequence of random variables (Bunow & Weiss, 1979) and the existence of these trajectories raises intersting philosophical and biological questions (May & Oster, 1976).

Does chaos occur in nature? Several authors (Hassel et al, 1976; Bellows, 1981) have addressed this question. The conclusion has emerged that natural populations are well damped and not susceptible to complex dynamics. These studies, however, are weighted toward temperate zone insets with a turnover time of one year. We hope to show that much stronger potential for complex dynamics exists for multivotine species in a seasonally fluctuating environment.

The logistic difference equation

$$N_{t+1} = (1+r)N_t - (r/K)N_t^2 \qquad \ldots\ldots\ldots (2.4.1)$$

(Maynard-Smith, 1968; May; 1972) is typical of many "chaotic" models. This equation maps population density at time t, N_t into N_{t+1}; r measures the potential for population increasing at zero density; and K is the carrying capacity of the environment. From a dynamical systems viewpoint, r is the important parameter inasmuch as it tunes the severity of the density

dependence and thus determines the asymptotic dynamics. K, on the other hand merely scales N. Accordingly, Eq(2.1) is often rescaled by letting

$$X_t=(r/[(1 + r)K]N_t \quad (2.2)$$

Equation(2.1) then takes on the simpler form

$$X_{t+1}= sX_t(1 - X_t), \quad (2.3)$$

where s = (1 + r)

As written, r and K have fixed values. This implies a constant environment. Many species, however, live in environments which are seasonal with regard to factors such as temperature and rainfall (Fretwell, 1972). For such species, one expects predicable changes in physiology and condition over the course of the year, and thus seasonal variation in r and K. For continuous systems, it has been shown that seasonal variation may affect dynamics (Rosenblat, 1980, Cohen & Rosenblat, 1982; Nisbet & Gurney, 1982). In general, periodic coefficients may lead to parametric resonance (Arnold, 1978; Nayfeh, 1979). Little, however, has been done with discrete models (but see May & Oster, 1976, Appendix C). In particular, whether seaonality favors higher order, cycles, and Chaos is a question that has not been addressed. To consider this question, we proceed as follows:

We develop a simple model of seasonality - two seasons (good,bad)-for species with two nonoverlapping generations per year.

This model has interesting features which may be examined analytically or geometrically. Some of the features appear quite robust. Their biological significance is discussed.

A SIMPLE MODEL OF SEASONAL GROWTH

Consider an insect with two discrete, nonoverlapping generations each year. The population is censused at the beginning of fall and spring. We assume that there exists a logistic map with one set of parameters to account for the event of summer and a similar map with different parameters for the winter. If N_ϕ and N_σ represent fall and spring densities, we then have

$$N_\phi = (1+r)N_\sigma - (r/K)N_\sigma^2 \quad \cdots (2.4.4a)$$

$$N_\sigma = (1+\tilde{r})N_\phi - (\tilde{r}/\tilde{K})N_\phi^2 \quad \cdots (2.4.4b)$$

For convenience let

$$N_s = [r/|(1+r)K|]N_\sigma \quad \cdots (2.4.5a)$$

$$N_f = (\tilde{r}/[(1+\tilde{r})\tilde{K}])N_\phi \quad \cdots (2.4.5b)$$

In terms of these new variables, Eqs (2.4.4a) & (2.4.4b) become

$$N_f = aN_s(1-N_s) \quad \cdots (2.4.6a)$$

$$N_s = bN_f(1-N_f) \quad \cdots (2.4.6b)$$

Here,

$$a = \frac{(1+r)^2\,\tilde{r}K}{(1+\tilde{r})\,r\tilde{K}} \quad (2.4.7a)$$

$$b = \frac{(1+\tilde{r})^2\,r\tilde{K}}{(1+r)\tilde{r}K} \quad (2.4.7b)$$

We also introduce new parameters, $\bar{s}$ and ε defined by

$$a = \bar{S}\frac{(1+\varepsilon)}{(1-\varepsilon)} \qquad \ldots\ldots (2.4.8a)$$

$$b = \bar{S}\frac{(1-\varepsilon)}{(1+\varepsilon)} \qquad \ldots\ldots (2.4.8b)$$

Note that $\bar{S}$ is the geometric mean of a and b, and that ε, which we take as our measure of seaonality, gauges the deviation of a and b from $\bar{S}$. As defined, ε lies between plus and minus one. Switching the sign of ε simply interchanges a and b. We take system (2.4.6) with a and b as in (2.4.8) as our fundamental system of equations.

The author has given numerical results and graphical analysis.

CHAPTER-III

ASSORTATIVE MATING MODELS

3.1 INTRODUCTION:

Assortative mating means that the mated pairs are more similar for some phenotypic trait than if they were chosen at random from the population. It may have either of two causes, or some combination of both. The tendency towards phenotypic similarity of mating pairs may be a direct consequence of genetic relationship. For example, in a subdivided population there will generally be a greater phenotypic similarity among the members of a subpopulation because they share a common ancestry. The genetic consequences in this case are the same as those of inbreeding.

On the other hand, there may be assortative mating based on similarity for some trait and any genetic relationship is solely a consequence of similar phenotypes. For example, there is a high correlation between husband and wife for height and intelligence, probably caused much more by non-random marriage associated with the traits themselves than by common ancestry.

There are also other situations. For example, there is a considerable correlation in arm length between husband and wife. This is probably a consequence of the fact that those factors, genetic and environmental, that increase height also increase the length of the arm. So, any assortative mating for height will be reflected in a assortative mating for arm length,

diminished somewhat by the lack of perfect correlation between the two traits.

Assortative mating is between individuals of similar phenotypes; inbreeding is between individuals of similar genotypes. Since individuals with similar phenotypes will usually be somewhat similar in their genotypes. We shall expect assortative mating to have generally the same consequences as inbreeding. An excess of consanguineous mating in a population has two effects. (1) an increase in the average homozygosity and (2) an increase in the total population variance. Assortative mating would be expected to produce the same general kinds of results.

This model is being discussed under two subheadings namely phenotypic and genotypic assortative matings.

3.2 PHENOTYPIC ASSORTATIVE MATING MODEL:

Consider a population of diploid individuals at a single locus with two alleles G and g with respective probabilities p and q. Hence after random mating, the probability of occurrence of three genotypes GG, Gg and gg will be p^2. $2pq$ and q^2 respectively. In this type of mating, the mating occurs between phenotypically similar individuals. Four different kinds of crosses are formed namely GG x GG, GG x Gg, Gg x Gg and gg x gg. If there is a complete selection against the homozygous recessives (gg), then the cross gg x gg does not exist and only the remaining three crosses are possible. Even then there are still recessive genes (g) present in the population with their dominant alleles (G) in the form of hetrozygotes (Gg) and the recessive

individuals (gg) are produced in every generation from Gg x Gg matings. The probability of occurrence of such type of individuals (gg) in the zeroth generation is q^2 and they are removed consequently. In the first generation, the homogygous recessive offsprings (gg) appear as a result of mating between heterozygotes (Gg x Gg) in which the one fourth of the ofsprings are expected to be homogygous recessives (gg). Along with the dominant phenotypes (GG and Gg), the probability of their occurrence will be $q^2/(1+q)^2$ and they are removed. Again after mating between heterogygotes (Gg x Gg), the probability of their occurrence (gg) in the second generation will be $q^2/(1+2q)^2$ and so on. An outline of this technique for obtaining the probability of selection against recessive genotypes (gg) is shown in table:

TABLE

PHENOTYPIC PROBABILITIES IN ASSORTATIVE MATING:

(AFTER REMOVAL OF RECESSIVES)

GENOTYPE	GG	Gg	gg	PROBABILITY OF SELECTION AGAINST RECESSIVE GENETYPES
Probability of occurrence in the zeroth generation	p^2	$2pq$	q^2	
Relative fitness	1	1	0	$p_o = q^2$
Genetypic ratio after selection	p^2	$2pq$	0	

Mating type

GG x GG	p^4	0	0
GG x Gg	$2p^3q$	$2p^3q$	0
Gg x Gg	p^2q^2	$2p^2q^2$	p^2q^2

Genotypic ratio in the first generation $p^2 \quad 2pq \quad q^2$

Probability of occurrence in the first generation $\frac{1}{(1+q)^2} \quad \frac{2q}{(1+q)^2} \quad \frac{q^2}{(1+q)^2}$

Relative fitness 1 1 0

$p_1 = \frac{q^2}{(1+q)^2}$

Genotypic ratio after selection $\frac{1}{(1+q)^2} \quad \frac{2q}{(1+q)^2} \quad 0$

Mating type

GG x GG	$\frac{1}{(1+q)^4}$	0	0
GG x Gg	$\frac{2q}{(1+q)^4}$	$\frac{2q}{(1+q)^4}$	0
Gg x Gg	$\frac{q^2}{(1+q)^4}$	$\frac{2q^2}{(1+q)^4}$	$\frac{q^2}{(1+q)^4}$

Genotypic ratio in the second generation $\frac{1}{(1+q)^2} \quad \frac{2q}{(1+q)^3} \quad \frac{q^2}{(1+q)^4}$

Probability of occurrence in the second generation	$\frac{(1+q)^2}{(1+2q)^2}$	$\frac{2q(1+q)}{(1+2q)^2}$	$\frac{q^2}{(1+2q)^2}$	
Relative fitness	1	1	0	$P_2 = \frac{q^2}{(1+2q)^2}$
Genotypic ratio after selection	$\frac{(1+q)^2}{(1+2q)^2}$	$\frac{2q(1+q)}{(1+2q)^2}$	0	

proceeding in this way we see that the probability of selection against recessive genotype (gg) in the nth generation is

$$p_n = \frac{q^2}{(1+nq)^2}; \quad n = 0, 1 \ldots . \qquad (3.2.1)$$

It is clear that the selection becomes decreasingly effective as the recessive gene becomes rare. Sooner or later it is to be expected that all the favourable alleles (G) originally segregating will be brought to fixation. As they approach fixation, the genetic variance declines and the rate of response diminishes, till, when fixation is complete, then response should cease. When the response has ceased, the population is said to be at the selection limit.

If the culling of recessive genotypes is considered as emigration, then the present situation of reproduction is explained by the model.

A. EXPECTED POPULATION SIZE WITH NON-RECESSIVE GENOTYPE:

Expected population size after culling the unwanted recessive genotypes (gg) can be obtained. Therefore, the expected population size at the nth generation is given by:

$$E(Z_n) = (1-q^2)\left(1-\left(\frac{q}{1+q}\right)^2\right)\left(1-\left(\frac{q}{1+2q}\right)^2\right)\cdots\cdots$$

$$\cdots\cdots\left(1-\left(\frac{q}{1+(n-1)q}\right)^2\right)\mu^n$$

$$= \mu^n \prod_{i=0}^{n-1}\left[1-\left(\frac{q}{1+iq}\right)^2\right],$$

$$n = 1, 2, \cdots \quad (3.2.2)$$

B. EXPECTED NUMBER REMOVED:

We see that the recessive genotypes are being removed as a result of culling in different generations. Their expected number upto any generation can be obtained by substituting in (3.2.2) the probability of selection from (3.2.1) Hence it is given by

$$\text{Expected number removed upto nth generation} = \sum_{k=0}^{n} \mu^k\left[\frac{q^2}{(1+kq)^2}\prod_{i=0}^{k-1}\left(1-\left(\frac{q}{1+iq}\right)^2\right)\right]$$

$$\cdots\cdots\cdots (3.2.3)$$

3.3 GENOTYPIC ASSORTATIVE MATING MODEL

This mating is different from the phenotypic - assortative mating as in the case of former,

the dominant homogygotes (GG) and heterozygotes (Gg) are distinct entities. In this system, the mating is allowed between the individuals of the same genotype. Hence only three different type of crosses namely GG x GG, Gg x Gg and gg x gg are possible whereas the cross GG x Gg which is permitted, in case phenotypic assortative mating, is not possible in this case. Even the third cross gg x gg will not exist as there is complete selection against the homozygous recessive genotype (gg). Therefore only the two crosses GG x GG and Gg x Gg will contribute to the progeny of the next generation.

In the zeroth generation, the probability of occurrence of homozygous recessives (gg) is q^2 and they are removed consequently. In the first generation homozygous recessives (gg) appear as a result of mating between heterozygotes (Gg x Gg). The probability of their occurrence is $q^2/(p^2+4q^2)$ and they are removed. In the 2nd generation, the probability of selection against homozygous recessives turns out to be

$$\frac{q^4}{(p^2+2q^2)^2+4q^4}$$

and so on in successive generations. An outline of the above technique is presented in the table given below:

GENOTYPIC PROBABILITIES IN ASSORTATIVE MATING (AFTER REMOVAL OF RECESSIVES)

GENOTYPE	GG	Gg	gg	PROBABILITY OF SELECTION AGAINST RECESSIVE GENOTYPES (gg)
Probability of Occurrence in zeroth generation	p^2	$2pq$	q^2	
Relative fitness	1	1	0	$p_0 = q^2$
Genotypic ratio after selection	p^2	$2pq$	0	
Mating type				
GG x GG	p^4	0	0	
Gg x Gg	p^2q^2	$2p^2q^2$	p^2q^2	
Genotypic ratio in the first generation	$p^2(p^2+q^2)$	$2p^2q^2$	p^2q^2	
Probability of occurrence in the first generation	$\frac{p^2+q^2}{p^2+4q^2}$	$\frac{2q^2}{p^2+4q^2}$	$\frac{q^2}{p^2+4q^2}$	
Relative fitness	1	1	0	$p_1 = \frac{q}{p^2+4q^2}$
Genotypic ratio after selection	$\frac{p^2+q^2}{p^2+4q^2}$	$\frac{2q^2}{p^2+4q^2}$	0	

Mating type

GG x GG $\frac{(p^2+q^2)^2}{(p^2+4q^2)^2}$ 0 0

Gg x Gg $\frac{q^4}{(p^2+4q^2)^2}$ $\frac{2q^4}{(p^2+4q^2)^2}$ $\frac{q^4}{(p^2+4q^2)^2}$

Genotypic ratio in the second generation $\frac{(p^2+q^2)^2+q^4}{(p^2+4q^2)^2}$ $\frac{2q^4}{(p^2+4q^2)^2}$ $\frac{q^4}{(p^2+4q^2)^2}$

Probability of occurrence in the second generation $\frac{(p^2+q^2)^2+q^4}{(p^2+q^2)^2+4q^4}$ $\frac{2q^4}{(p^2+q^2)^2+4q^4}$ $\frac{q^4}{(p^2+q^2)^2+4q^4}$

Relative fitness 1 1 0 $P_2 = \frac{q^4}{(p^2+q^2)^2+4q^4}$

Genotypic ratio after selection $\frac{(p^2+q^2)^2+q^4}{(p^2+q^2)^2+4q^4}$ $\frac{2q^4}{(p^2+q^2)^2+4q^4}$ 0

Proceding in this way, we see that the probability of selection against unwanted recessive genotypes (gg) in different generations is as follows:

$$P_o = q^2$$

$$P_1 = \frac{q^2}{p^2+4q^2}$$

$$P_2 = \frac{q^4}{(p^2+q^2)^2+4q^4}$$

$$P_3 = \frac{q^8}{[(p^2+q^2)^2+q^4]^2+4q^8}$$

and so on.

In general

$$p_n = \frac{q^{2^n}}{\left[\left(\left(\left(\left(p^2\right)^{2^0} + q^{2^1}\right)^2 + q^{2^2}\right)^2 + q^{2^3}\right)^2 + \cdots + q^{2^{n-1}}\right]^2 + 4q^{2^n}};$$

$$n = 1, 2, \ldots \quad \ldots\ldots\ldots (3.3.1)$$

An approximation for general p_n can be done through a power series.

3.4. CONTINUOUS - GENOTYPE MODELS AND ASSORTATIVE MATING:

In reference to the above Genotypic Assortative Model a careful consideration is done by Feldman and Cavalli-Sforza. Consideration of the models of Wright, Fisher, Bulmer, and Lande in the context of assortative mating verses mutation shows that these models are consistent with each other. This note is an attempt to address some of the questions, in particular whether the classical theory of assortative mating due to Fisher (1918) and Wright (1921) involves arbitrary and unjustified assumptions. Clarification of the above establishes clearly the connection between Wright's (1921) classical treatment of assortative mating and the more recent continuous -Genotype quantitative character models of Bulmer (1971) and Lande (1975). All of these models take linkage disequilibrium into account correctly. This treatment of likage disequilibrium seems to be a source of the discrepancy between the results of these authors and those of Feldman and Cavalli-Sforza.

CONVERGENCE PROBLEM IN ASSORTATIVE MATING

Feldman and Cavalli-Sforza (1979a) consider, as do all author, a quantitative character which is sum of individual locus effects at a number of loci, plus an environmental effect which is independent of the Genotype (for the purposes of this argument we concentrate as did Feldman and Caralli-Sfroza in the relevant section of their work, on cases lacking cultural transmission). They declare the previous treatments of Fisher (1918) and Wright (1921) to "[use] concepts of inbreeding at a single locus extended in a heuristic way to many loci". They cite the classical equilibrium result

$$\hat{V}_p = V_A/(1- r) + V_D + V_E \ , \qquad (3.4.1)$$

where V_A, V_D, and V_E are the usual additive, dominanace, and environmental components of variance in the initial generation, which is assumed to have been in Hardy-Weinberg Proportions and at linkage equilibrium. In(3.4.1) the quantity r is the correlation "between the genotypes", that is, between the breeding values of mated pairs. They then ask "of what system is [Eq(3.4.1)] an equilibrium?". They illustrate their problem by deriving reccurrence of genetic variation under assortative mating in their quantitative character model. That model is (Feldman and Cavalli-Sforza, 1979 a. Eq 17)

$$g_{t+1} = (g_t + g'_t)/2 + x_t \ , \qquad (3.4.2)$$

where g_t and g'_t are random variables giving the breeding values of the two Parents of an individual ,

x_t is a within-family random variable expressing the effects of genetic segregation within the family and g_{t+1} is the breeding value of the resulting offspring. In their development, g_t and g'_t are of course correlated as a result of the assortative mating. The segregation variable x_t is uncorrelated with g_t and g'_t. A key feature of Feldman and Cavalli-Sforza'a system is that the variance of x_t is taken to be equal to $G_t/2$, where G_t is the variance of g_t. Based on(3.4.2) and this assumption, they obtain a recursion for G_t under the assumption that the correlation between breeding values is a by-Product of a correlation between phenotypes. They show that this recursion implies that $G_t \longrightarrow \infty$ if r_{pp}, the phenotypic correlation of mates, is a positive constant. This lack of convergence is alarming in that it raises the possibility that the equilibrium(3.4.1) may not be achieved.

WRIGHT'S RECURSION AND THEIR IMPLICATIONS

The two classical treatments of assortative mating reach completely compatible conclusions, so it seems resonable to confine our attention to Wright's (1921) treatment, which is easier to follow. As we will see, Wright's argument involves a limited and straight forward set of assumptions and is a dynamic model, not requiring any extra assumptions to bring about equilibrium. Moran and Smith (1966) have reprinted Fisher's (1918) papers with an detailed commentary on his methods. Kempthorne (1957, Chap 22) present an extensive rederivation of Fisher's assortative mating results, with much additional detailed, and Malecot's celebrated 1939 thesis (reprinted in Malecot, 1966 makes explicit many of the points implicits in Fisher's paper, fills in many steps in the argument

and makes some extensions.

Wright assumes a discrete-generation, mon ecious, diploid population of infinite size. The character is the sum of effects at n loci, each locus having two alleles. There is no dominance, so that the Phenotypic variance will be the um $V_A + V_E$, without dominance variance. The loci have equal effects (are exchangeable), so that the effects of the A Allele on the Phenotype is the same as the effect of B. The effect of substituting allele A for a (or B for b) is α. Initially all loci are the linkage equilibrium with equal gene frequencies (p) of alleles A,B,C ....and frequencies 1-P of alleles a, b, c ...This symmet-ry of gene frequencies as maintained throught Wright's analysis. The environmental effect is independent of all genetic effects, and has a variance of V_E. Hereafter we use G_t and E instead of V_A and V_E, for compatibility with Feldman and Cavalli-Sforza's notation.

Assortative mating is assumed to make place in such a way that all individuals have equal expected numbers of offspring, so that the gene frequencies will not change. Assortative mating is assumed to create a correlation, γ_{pp}, between phenotypes with the probability of two genotypes mating being conditionally independent give their phenotypes Karlin (1979a) has called particular attention to the conditional independence assumption implicit in the classical (and subsequent) treatments of assortative mating. The assumption of a given correlation of phenotypes, together with their conditional independence of genotypes is sufficient to allow determination of co-variance between allele effects in the population in most simple

relationships, but when double first cousins are considered, one must specify more details of the mechanism of asortative mating. It was presumably for this reasons that Nagylaki 1978 found himself unable to extend his analysis to double first cousins.

Using these assumptions, Wright (1921) was able to obtain recurrence equation for various correlation between effects of different genes, without any consideration of higher moments of the joint distribution of gene effects or of higher moments of the joint distribution of phenotype of mates. His argument has been translated into variances and co-variances by crow and Felsenbtein (1968; also crow and Kimura, 1970, pp 148-152), and the present section will be in those terms.

Consider the system of correlations of gene effects in two mates and their offspring. The combination of the exchangeability of loci in the initial population and the conditional independence of genotypes given phenotypes, allow the correlations of gene effects in an individual of generation t to be reduced to three quantities.

f_t= the correlation of effects of alleles at the same locus,

k_t= the correlation of effects of non-homologous loci in the same gamete,

and

l_t= the correlation of effects of non-homologous loci in opposite gametes,
and the correlation of gene effects between mates reduce to two quantities,

m_t= correlation of effects of alleles at the same locus in mates

m'_t= correlation of effects of alleles at different loci in mates.

Symmetry considerations then require that $m_t = m'_t$. Since $\ell_{t+1}=m'_t$ and $f_{t+1} = m_t$, we then also have that $f_{t+1}=\ell_{t+1}$.

An imporant assumption of Wright's is that all n loci are unlinked. Given that

$$K_{t+1} = \tfrac{1}{2}K_t + \tfrac{1}{2}\ell_t \qquad (3.4.3)$$

(Incidently, one must note here that the statement of crow and Felsenstein (1968) that can analyze this system in terms of a mean recombination fraction $\bar{c}$ is erroneous. All loci must be unlinked for this analysis to work, although using $\bar{c}$ does yeild the correct equilibrium of the system). Equation(3.4.3) is one of two dynamical equations of Wright's argument. The other is obtained from a calculation of the genetic variance by adding up the single gene variances and all the covariance:

$$G_t = pq\alpha^2[2n + 2nf_t + 2n(n-1)K_t + 2n(n-1)\ell_t]$$
$$= 2npq\alpha^2[1+(n-1)K_t + n\ell_t], \quad \ldots\ldots (3.4.4)$$

which, together with the expression for the additive genetic covariance of mates,

$$C_{AA'}^{(t)} = 4n^2 m_t pq\alpha^2 \qquad \ldots\ldots\ldots (3.4.5)$$

and the requirement that $C_{AA'}^{(t)}/G_t = r_{gg}^{(t)}$ (the correlation of genotypes in generation t), yields

$$m_t = \frac{r_{gg}^{(t)}}{2n}[1+(n-1)K_t + n\ell_1] \quad \ldots\ldots (3.4.6)$$

so that since $\ell_{t+1} = m_t$,

$$\ell_{t+1} = \frac{r_{gg}^{(t)}}{2n}[1+(n-1)K_t + n\ell_t] \quad \ldots (3.4.7)$$

Equation(3.4.3) and(3.4.7) are a full dynamic recursion system (within the limits of the exchangeability assumption). We have not yet determined $r_{gg}^{(t)}$, but

this is easily done in the same way that Feldman and Cavalli-Storza do in their recursions:

$$r_{gg}^{(t)} = r_{pp}\left(\frac{G_t}{G_t + E}\right) \qquad \ldots\ldots (3.4.8)$$

one can go on from (3.4.8), (3.4.4), (3.4.7) and (3.4.3) to examine convergence of the quanlities ℓ_t and K_t to equilibrium, and show that Wright's version of (3.4.1).

$$\hat{V}_p = \frac{V_A}{1-(1-1/2n)r} + V_E \qquad \ldots\ldots (3.4.9a)$$

or in our notation if initially $K_t = \ell_t = f_t = 0$,

$$\hat{G}_1 = \frac{G_0}{1-(1-1/2n)\hat{r}_{gg}} + E \qquad \ldots\ldots (3.4.9b)$$

is the only equilibrium possible unless $r_{pp} = 1$ and $E = 0$. Wright's analysis was based on a fully defined (if not formally stated) dynamical system of recurrence equations, (see Wright, 1921, p.148) and does not involve any arbitrary assumption of equilibrium..

Wright's model is, however, not a comprehensive treatment of assortative mating. It makes number of symmetry assumption, particularly the exchangeability of loci. This assumption forces symmetry of locus effects of gene frequencies, and of the linkage map (independence of all loci). The analysis in terms of f_t, k_t, ℓ_t and m_t implicity assumes an initial population configuration in which the pattern of covariance among individual locus effect also show exchangeability of loci. A fully analysis of multiple locus assortative mating would be desirble. Nevertheless, the result of such an analysis would have to be consistent with Wright's result, unless it could somehow be shown that his equilibria are found to be unstable when departures from symmetry are allowed.

RECURSIONS OF VARIANCE COMPONENTS

Instead of working thought the details of Wright's argument, it will be more instructive to take as our dynamic variable certain components of variance. In terms of Wright's variables f_t, k_t and ℓ_t the total genetic variance is

$$G_t = 2npq\alpha^2(1 + f_t + (n-1)k_t + (n-1)\ell_t) \quad \ldots\ldots (3.4.10)$$

The initial genetic variance of the population (which initially has $f_t = k_t = \ell_t = 0$) is

$$V_o = 2npq\alpha^2 \quad \ldots\ldots (3.4.11)$$

We can break the variance (3.4.10) into two parts

$$G_t = V_o(1 + (n-1)k_t) + V_o(f_t + (n-1)\ell_t)$$
$$= V_o(1 + (n-1)k_t) + V_o(n\ell_t), \quad \ldots (3.4.12)$$

since, as we have seen, $f_t = \ell_t$. The two terms on the right side of (3.4.12) represent the parts of the variance which come from variance within a single gamete (either from variance of individual gene effects or from co-variance between effects of genes which came from the same parent), and covariance between different gametes as a result of assortative mating. Let us denote these as respectively, K_t amd L_t :

$$K_t = V_o(1 + (n-1)k_t), \quad \ldots\ldots (3.4.13a)$$
$$L_t = V_o\, n\ell_t \quad \ldots\ldots (3.4.13b)$$

we can solve these for k_t and ℓ_t:

$$k_t = (K_t - V_o)/[(n-1)V_o], \quad \ldots\ldots (3.4.14a)$$
$$\ell_t = (L_t/nV_o) \quad \ldots\ldots (3.4.14b)$$

Substituting these into Wright's recursions and

eleminating k and ℓ, we obtain from (3.4.3)

$$K_{t+1} = \frac{1}{2} V_0 + \frac{1}{2} K_t + \frac{1}{2}\left(1 - \frac{1}{n}\right) L_t \quad \ldots (3.4.15a)$$

and from 7) after some algebra

$$L_{t+1} = \frac{r_{gg}^{(t)}}{2} K_t + \frac{r_{gg}^{(t)}}{2} L_t , \quad \ldots\ldots (3.4.15b)$$

If r_{gg} were constant, Eqs.(3.4.15a) and(3.4.15b) would be linear. However, since we are interested in case in which r_{pp} is held constant, we must add, from (3.4.8),

$$r_{gg}^{(t)} = r_{pp} \frac{(K_t + L_t)}{(K_t + L_t + E)} \quad \ldots\ldots (3.4.15c)$$

This makes the recursion system(3.4.15) a non-linear one whose global stability properties are not easily analyzed. Numerical iteration of(3.4.15) for a variety of cases has, in hands, failed to find any case in which the equilibrium is not globally stable.

One can use Wright's argument to obtain an expression for the within-sibship variance. Given any two particular parents each gamete transmitted to the offspring contains one of the two copies at each of the n loci, chosen randomly and independently under thse conditions the genetic variance of the offspring in a sibship, around their mean, has the expected value

$$2npq\alpha^2 \frac{1}{2}(1 - f_t) = \frac{1}{2} V_0 (1 - f_t) \quad \ldots\ldots (3.4.16)$$

The phenotypic variance within sibship is increased over this value by the environmental variance E (in a more realistic model by that part of the environmental variance not attributable to effects common to all sibs).

The quantity f_t is equal to 1_t. Equations (3.4.13b) and(3.4.15b) show that this is $r_{gg}/(2n)$ times G_t/G_o. The within-sibship genetic variance thus

remains nearly constant, being reduced slightly by a fraction of order 1/n. Wright's model thus involves no assumption of exact constancy of within-sibship variance, but predicts a slight decline in this quantity, a decline which can be ignored if n is large.

RECURSIONS WITH INFINITELY MANY LOCI

Take the recursion system (3.4.15), let $n \to \infty$, and then add Eqs. (3.4.15a) and (3.4.15b), we have

$$K_{t+1} + L_{t+1} = \tfrac{1}{2}(1 + r_{gg}^{(t)})(K_t + L_t) + \tfrac{1}{2} V_0 \quad \ldots\ldots (3.4.17)$$

Note that (3.4.17) and (3.4.15c) contain K_t and L_t only as their sum $K_t + L_t$, (which is the total additive genetic variance G_t. Substituting, we have the recursion in G_t:

$$G_{t+1} = \tfrac{1}{2}(1 + r_{gg}^{(t)}) G_t + \tfrac{1}{2} V_0 \,, \quad \ldots\ldots\ldots (3.4.18)$$

and from (3.4.15c).

$$G_{t+1} = \tfrac{1}{2}\left[1 + r_{pp} G_t / (G_t + E)\right] G_t + \tfrac{1}{2} V_0 \quad \ldots (3.4.19)$$

This is the limit of the recursion system (3.4.15) as $n \longrightarrow \infty$, but this would not necessarily guarantee that the equilibrium of this recursion will be the limit of the equilibrium of (3.4.15) as $n \to \infty$. This is, however, the case, as can be seen if one compares numerical results. The equilibrium of (3.4.19) is a solution of the quadratic

$$G^2(1 - r_{pp}) + G(E - V_0) - V_0 E = 0 \quad \ldots\ldots (3.4.20)$$

(in practice, the larger of the two solutions of the quadratic seems the relevant one). Alternatively we can obtain from (3.4.18)

$$G = V_0 / (1 - r_{gg}) \,, \quad \ldots\ldots\ldots\ldots\ldots (3.4.21a)$$

where

$$r_{gg} = \left[G / (G + E)\right] r_{pp} \quad \ldots\ldots (3.4.21b)$$

and substituting(3.4.21a) into(3.4.21b) we have the following quadratic equation for the equilibrium value of r_{gg}:

$$r_{gg}^2 E - r_{gg}(V_0 + E) + r_{pp} V_0 \quad \ldots\ldots\ldots\ldots (3.4.22)$$

The recursion (3.4.19) is described by Feldman and Cavalli-Sforza as having a unique positive equillibrium which is globally stable, as long as $r_{pp} < 1$. When $r_{pp} = 1$, it seems well behaved except when $E = 0$, in which case $r_{gg} \equiv 1$ so that $G_t \to \infty$ as $t \to \infty$.

RELATION TO FISHER's AND WRIGHT's SOLUTIONS

It is seen that(3.4.18) may be obtained from Wright's recursion by letting $n \to \infty$. The equilibrium obtained from(3.4.20), or equivalently from(3.4.21) and (3.4.22), is precisely Fisher's equilibrium solution. Equation(3.4.22), if divided by $V_c + E$, can be seen to be the same as Fisher's quadratic equation (crow and Kimura, 1970, p. 157). This is also the limit of Wright's equilibrium solution as $n \to \infty$. In the first generation of the recursion, if $G_o = V_o$, we obtain from(3.4.19).

$$G_1 = V_0 (1 + r_{pp} H/2), \quad \ldots\ldots\ldots\ldots (3.4.23).$$

Where $H = G_0/(G_{T_0} + E)$ is the initial heritability. This shows that, contrary to Feldman and Cavalli-Sfroza's implication (1979, pp.285-286) both the eqquilibrium solution and the first generation transitions of Wright's and Fisher's model can be obtained from a well specified dynamic system.

Feldman and Cavalli-Sforza (1977,p.171) describe to Fisher (1918) the position that the within-sibship variance is $G_t/2$ under assortative mating. However, Fisher's argument, as described by these a thors, was that assortative mating had little effect on the initial value Go/2. This interpretation on their parts seems to be the crucial difference between the position they ascribe to Fisher and the one is ascribed to him. Feldman and Cavalli-Storza (1979a) have emphasized the

crucial nature of the within-sibship variance in this argument.

Relation to Bulmer's Recursion

Equation(3.4.18) and(3.4.19) are also completely consistent with the continuous - genotype model of Bulmer (1971). Bulmer considered a model with a large number of loci, each of infinitesimal effect, as did Fisher (1918). By means of an indirect argument involving offspring - parent regressions, Bulmer suggested that natural selection which resulted in a change of genetic variance would act primarily by creating linkage disequilibrium. Since the loci were all assumed by Bulmer to be unlinked, half of this linkage diequilibrium will decay each genertion, so that the genetic variance G_t moves half-way back to its initial value each generation. Bulmer's argument applies to changes due to natural selection, and presumes random mating. A version of his argument which allows for correlation between mates leads to Eq(3.4.18).

THE CLASSIFICATION OF CONTINUOUS - GENOTYPE MODELS.

It is so far demonstrated that a full dynamic treatment of variances and co-variances, if taken to the limit of an infinite number of loci, behaves as if the within-sibship variance remains constant at $G_0/2$, although no specific assumption to this effect need be made. Some light can be shed on Feldman and Cavalli-Sforza'a procedure if we turn to the case of a balance between normalizing selection and mutation, for these the issues are basically the same. We find that all existing continuous-genotype models fall into one of three families. One contains the model of Fisher's

(1918), Wright (1921), Bulmer (1971), and Lande (1975). The second contains the model of Slatkin (1970) and Karlin (1929a-d). The second contains the model of Cavalli-Sforza and Feldman (1976, 1970, Feldman and Cavalli-Sfroza 1977, 1979a and 1979b)

LANDE's MODEL AND ITS ANTECEDENTS

Lande (1975) made a model within Loci, in which it is assumed that the joint distribution of additive effects at the loci remains multivariate normal under the operation of natural selection, recombination, and mutation. While this is at best an approximation Fleming (1979) has identified cases where it will be accurate, after an extensive analysis by Perturbation methods. The advantage of Lande's approach is that it approximates at least 2^n-n-1 linkage disequilibrium Parameters among n loci by only n(n-1)/2 covariances. Lande has solved explicitely for the equilibrium variances and covariances among Loci in the case where normalizing selection is opposed by mutation.

Lande's methods still require that many quantities be followed simultaneously. It has been presented elsewhere (1979) a symmetrized version of Lande's equations which greatly reduce the number of variables. If it is considered the subcase of Lande's model which has n Loci which are completely exchangeable and all unlinked, the n means, n variances, and n(n-1)/2 covariances reduce to three quantities, m, v, and c. We use these in the form of Phenotypic mean M= 2 nm, the part of the genotypic variance due to single gene effects. V = 2nv, and the remainder of the genotypic variance which is due to linkage disequilibrium, C= 2n(n-1)c. In the case of mutation Vs. stabilizing selection,

there are four parameters: U, the total increment of variance from mutations, E, the nonadditive variance of the phenotype, P, the optimum phenotype, and, S,

, the selection parameter. The latter two relect the fitness function

$$w(X) = \exp\left[-(X-P)^2/(2S)\right], \quad \ldots\ldots(3.4.24)$$

where X is the phenotype.

By a tedious argument (Felsenstien 1979) which may be ontained by symmetrizing Lande's (1975) equation, we find the following recursion for V and C (since these two are autonomous we ignore M):

$$V_{t+1} = V_t - \frac{1}{2n}\frac{(V_t+C_t)^2}{(V_t+C_t+S+E)} + U, \quad \ldots\ldots(3.4.25a)$$

$$C_{t+1} = \frac{1}{2}C_t - \frac{1}{2}\left(1-\frac{1}{n}\right)\frac{(V_t+C_t)^2}{(V_t+C_t+S+E)} \quad \ldots\ldots(3.4.25b)$$

when these are solved for V and C at equilibrium, the results are

$$\hat{C} = -2(n-1)U \quad \ldots\ldots\ldots\ldots (3.4.26a)$$

and

$$\hat{V} = (3n-2)U + \left[n^2U^2 + 2nU(S+E)\right]^{1/2} \quad \ldots\ldots(3.4.26b)$$

yielding a total equilibrium genetic variance

$$\hat{V}+\hat{C} = nU + \left[n^2U^2 + 2nU(S+E)\right]^{1/2} \quad \ldots\ldots(3.4.26c)$$

This is of course the same result which would be obtained by symmetrizing Lande's equilibrium solution.

If we take the recursion system (3.4.25) and let $n \to \infty$, we obtain Bulmer's results provided that

we let $U = o$. Then(3.4.25) consist of

$$V_{t+1} = V_t = V_o \quad \cdots\cdots\cdots (3.4.27a)$$

and

$$G_{t+1} = \frac{1}{2} G_t - \frac{1}{2} \frac{G_t^2}{(G_t + S + E)} \quad \ldots (3.4.27b)$$

where G_t is the total genetic variance $V_o + G_t$.
Careful consideration of Bulmer's (1971) Eq(3.4.18) will show it to be identical to Eq(3.4.27) Adding Eqs(3.4.27) we obtain

$$G_{t+1} = \frac{1}{2} G_t + \frac{1}{2} V_o - \frac{1}{2} \frac{G_t^2}{(G_t + S + E)} \cdot \quad \cdots\cdots (3.4.28)$$

It should thus be clear that Lande's result include Bulmer's method as a limiting special case. Like Wright and Fisher, both attempt an explicit look keeping of linkage disequilibrium effects. The first modern continuous genotype model was the treatment of the balance between mutation and normalizing selection by Kimura (1965), who used a one conditions time model. For weak selection ($S > o$), which is the only case for which it is proper to compare Kimura's continuous-time model to Lande's disrelative model, both yield an expected genetic variance $\sqrt{(2US)}$ .Wright and Fisher did not treat the balance between normalizing selection and mutation by the present mothods (though Wright did treat normalizing selection by other means). For the case of Assortative mating, where second moments are sufficient to describe the outcome we have seen that they obtained result concordant with Bulmer, Lande(1977) treated a combination of normalizing selection, mutation, and assortative mating. Finding that assortative mating effects the rate of approach to equilibrium but not the equilibrium genetic variance. Feldman and Cavalli - Sforza (1977) have treated a similar case by their methods which yeild different results.

"PHENOTYPIC EVOLUTION MODELS"

An alternative approach, due to Slatkin (1970) and expanded by Slatkin and Lande (1976) and Karlin (1979a-d) usually takes the offspring of a mating to be normally distributed around the midparent phenotype, with a constant variance V_k. This model is quite distinct from the other models mentioned so far. In these models, the mean of the offspring of a pair is the midparent genotypic mean $(g + g')/2$, whereas in phenotypic models it is the midparent phenotype $(p+p')/2$. Karlin (1979a-d) has made a large number of extension and generalization of this class of model to cover asymmetric transmission and multivariate phenotypes.

As long as it is not maintained that a phenotypic model represent any specific genetic situation, there can be little objection to using it. However, if it is represented as a more general model than an explicity genetic model this may be misleading Genetic models such as Lande's, Bulmer's, or Feldman and Cavalli - Sforza's are not specific case of existing phenotypic model

Cavalli -Sforza and Feldman's Model

In several of their papers (e.g. 1976,1976), these authors have treated the case of an equilibrium between mutation and normalizing selection. There equation for this case are, in the present notation and at the life stage corresponding to Lande's solution, before selection and recombination

$$G_{t+1} = G_t - \frac{G_t^2}{G_t + E + S} + U, \quad \ldots\ldots (3.4.29)$$

This differs from (3.4.28) in not averaging the genetic variance with V_o each generation. This amounts

to the presumption that all of the change of genetic variance wrought by selection goes into changing individual gene variances (changing the frequencies of alleles) and none into creating linkage disequilibrium or correlation between nonalleles on different gemetes. In the Bulmer-Lande approach, much of the change of genetic variance is taken up in this way. This was apparently for this reason that Cavalli - S forza and Feldman (1978, p.396) commented that " as we emphasized in our earlier work the component of variance due to segregation, $G_t^*/2$, is predicted on the basis of Hardy-Weinberg equilibrium; under selection or linkage dis-equilibrium, it may be inaccurate" Karlin (1979a p.339) comments that "this tactic seems quite arbitrary, apparently paraphrasing the presumption of additive equal allelic independent loci effects and global linkage equilibrium".

In fact both normalizing selection and assortative mating produce linkage disequilibrium, as well as correlation between genes on different genetes the effects of Feldman and Cavalli - S-forza's approximation in the assortative mating case were dramatic: failure of convergence of the genetic variance. In the case of the balance between mutation and normalizing selection the effects are also large. The solution of (3.4.29) a variant of Cavalli - S forza and Feldman's (1975) solution is

$$\hat{G} = \tfrac{1}{2} U + \tfrac{1}{2} \left[U^2 + 4U(E+S) \right]^{1/2} \quad \ldots\ldots (3.4.30)$$

Comparison of this with (3.4.26) will show agreement only when $n = \frac{1}{2}$, which is not a possible case. This apparently reflects the fact that even for only a single locus, their procedure does not acount for correlation induced by selection between the two homologous genes. When n is large and selection weak ($S > V$), their solution becomes approximation made by Cavalli-S Forza and Feldman leads to a discrepancy of a factor of $\sqrt{(2n)}$.

ENVOI

In thus seems that the model of Asher, Wright Kimud, Bulmer, and Lande form a reasonably self-consistent grouping and achieve an exact (Fisher and Wright) or approximate (Bulmer and Lande) accounting for linkage disequilibrium and other correlations. By contrast it is the model of Cavalli - Sforza and Feldman which is properly described as being a haploid single-locus model "extended in a heuristic way to many Loci".

A careful review of all of these models, of their assumption and interconnections, can only leave us admiring the preceptiveness of Wright and Fisher in extracting many of the most essential biologically relevant result while avoiding pit -falls. Admittedly, their models were not stated in extenso using the machinery and notation of mathematics. But that was a different era, one in which there was some common experience, and common assumption, shared between theoretician and experimental biologist. It was also an era in which the emphasis was on studying biologically relevant cases - which is what they did. And with what economy of notation, of space, of thought.

CHAPTER-IV

PREDATOR - PREY MODELS

4.1 INTRODUCTION:

There are variety of biological problems involving some kind of interaction between two or more species, such as competition between two species for a limited food supply or a prey-predator relationship in which one species is part of the food supply of the second species. The classical work on such problems, entailing a deterministic analysis, is largely due to Lotka (1925) and Volterra (1931). The phenomena included in studies of this type are often referred to under the heading of "THE STRUGGLE FOR EXISTENCE".

While a good deal can be done along deterministic lines, our problem is to inquire about the properties of the corresponding stochastic models. The transition probabilities are typically non-linear functions of the population sizes, and all the difficulties already encountered in epidemic theory are present with additional complications. Comparatively little has been done so far by way of a full stochastic investigation. However, some progress has been made (see Bertlett, 1960, Chapter 4 and 5, for a more detailed account) by starting with a deterministic model and trying to see what modifications would be required in the results if certain probability aspects were introduced at those points where they would be most likely to have an appreciable effect. Such speculative and heuristic methods may be very useful at an early stage in a difficult stochastic treatment, as they may indicate what kind of solutions we should look for and what kind of analytic

approximations might be appropriate.

4.2 A PREY-PREDATOR MODEL:

It is the situation where one species is at least part of the food supply of the other. We then have a Prey-Predator relationship. The first species can be regarded as the Prey or host, and the second as the Predator or Parasite according to circumstances.

The simplest deterministic model of the Lotka-Volterra type is based on the frequency of encounter between members of the two species. And it is reasonable, as a first approximation, to take this frequency as proportional to the population sizes. Thus in time Δt we can suppose that the first species gains an amount $\lambda_1 H \Delta t$ due to new birth and loses an amount $\mu_1 HP \Delta t$ due to predation. Similarly, if the second species has a birth rate proportional to the number of Prey available we can take its gain in Δt as $\lambda_2 HP \Delta t$, and its loss due to death as $\mu_2 P \Delta t$. The appropriate differential equation are thus

$$\left.\begin{aligned} \frac{dH}{dt} &= \lambda_1 H - \mu_1 HP \\ \frac{dP}{dt} &= \lambda_2 HP - \mu_2 P \end{aligned}\right\} \quad \ldots\ldots\ldots\ldots (4.2.1)$$

This time there is an equilibium point given by

$$(H,P) = (u_2/\lambda_2, \lambda_1/\mu_1) \quad \ldots\ldots\ldots (4.2.2)$$

It can be shown that this equilibrium point is neutral in the sense that there is no damping towards it for Population Point at any other part of the (H,P) plane. Thus division of the two equation given in (4.2.1) gives

$$\frac{dH}{dP} = \frac{(\lambda_1 - \mu_1 P) H}{(\lambda_2 H - \mu_2) P} \qquad \ldots\ldots\ldots (4.2.3)$$

which integrates to give

$$f(H,P) = -\mu_2 \log H + \lambda_2 H - \lambda_1 \log P + \mu_1 P = \text{constant} \qquad \ldots\ldots (4.2.4)$$

The solution is therefore represented by the set of closed curves given by f (H,P) = Constant, for different values of the constant.

For small curves around the equilibrium point it can be written :

$$H = \frac{\mu_2}{\lambda_2}(1+\xi), \quad P = \frac{\lambda_1}{\mu_1}(1+\eta) \ldots (4.2.5)$$

where ξ and η are small. Retaining only second order terms in ξ and η . We see that the paths are approximately the ellipses

$$\mu_2 \xi^2 + \lambda_1 \eta^2 = \text{constant} \qquad \ldots\ldots (4.2.6)$$

If larger curves are considered, it is clear that they will tend to be deformed from the elliptical shape by the Presence of the two axes.

Let us now imagine how these result would be modified by the introduction of suitable probability elements. If the equilibrium point is sufficiently far from each axis, if μ_2/λ_2 and λ_1/μ_1 are large enough. The deterministic model would be reasonably satisfactory for paths on which H and P remains fairly large. But if either of these quantities became small enough for statistical

fluctuations to be appreciable, a chance variation might cause the population point (H,P) to strike one of the axes, which are of course absorbing barriers,. Hence a stochastic model will be inherently unstable, because sooner or later the stochastic drift will cause one of the species to die out. The latter may be the Predator itself; or the Prey, in which case the Predator will die from lack of food.

Although the final extinction after only one or two cycle of the population point about the equilibrium value may be quite small. An exact analysis of this situation is not available, but we can undertake an approximate discussion as follows:

Consider the deterministic curves $f(X_1,X_2)=$ Constant. Elementary examination of the equation especially with regard to signs of the differential coefficients, shows that these curves are transversed in an anti-clockwise sense. Let us now look at the behaviour in the neighbourhood of the equilbrium point. Substituting ( ) in ( ) gives

$$\frac{d\xi}{dt} = -\lambda_1 \eta, \qquad \frac{d\eta}{dt} = \mu_2 \xi,$$

for small ξ and η. These equation imply

$$\frac{d^2\xi}{dt^2} = -\lambda_1 \mu_2 \xi \qquad \ldots\ldots (4.2.7)$$

It immediately follows that the motion is periodic with Period.

$$T = 2\pi(\lambda_1 \mu_2)^{-1/2}$$

Let us now suppose the Prey is fairly abundant so that the chance of extinction in a stochastic modification of the deterministic model is small. This means that we regard the equilibrium value of H, namely μ_2/λ_2 as large enough for probability effect near (μ_2/λ_2 μ_1/λ_1) to be negligible so as far as the first species is concerned. If however, P is only of moderate size, statistical fluctuations may well be important. They will have their greatest effect when P has its smallest value, P say, at the point of the relevant elliptical path nearest to the H axis. At the latter in the second equation of the (4.2.1) shows that dP/ dt=0. The effective birth and death rates per individual in the second population, namely $\lambda_2 H$ and u_2, are thus both equal at the point in question. But H is increasing and so we may write the birth-rate of P as $u_2 + \gamma \sin(2\pi t/T)$; where T is the period $2\pi(\lambda_1\mu_2)^{-\frac{1}{2}}$ is measured from the time of occurrence of the smallest value of P ; and the amplitude depends on the actual path being followed.

Thus in the neighbourhood of P' we can regard the second population as being approximately subject to a birth-and-death process with Birth-and-rates $\mu_2+\gamma\sin(2\pi t/T)$ and μ_2, respectively. This is a non-homogeneous process. We have, from Section 9.3 (Beiley), stochastic processes:

$$\begin{aligned} P(t) &= \int_0^t \{\mu(\tau) - \lambda(\tau)\}\, d\tau \\ &= -\int_0^t \gamma \sin(2\pi\tau/T)\, d\tau \\ &= \frac{\gamma T}{2\pi}\left(\cos\frac{2\pi t}{T} - 1\right) \quad \ldots\ldots (4.2.8) \end{aligned}$$

The chance of extinction, can thus be written as

$$P_0(t) = \left(\frac{J}{1+J}\right)^P \quad \ldots\ldots (4.2.9)$$

where P' is the smallest value of P, and

$$J = \int_0^t \mu_2 e^{\rho(\tau)} d\tau \quad \text{-----(4.2.10)}$$

The integral in (4.2.10) can be evaluated for any t, but we are especially interested in the region where t is of order $\frac{1}{4}T$. A comparatively simple solution exist for t = T, and we should not underestimate the value at t = $\frac{1}{4}T$ if we used t = T instead because the chance of extinction is always increases with t. In any case we are mainly interested in order of magnitude in discussions of the present type. When t = T, the integral in

$$J(T) = \int_0^T \mu_2 e^{\rho(\tau)} d\tau$$

$$= \mu_2 T e^{-\gamma T/2\pi} I_0(\gamma T/2\pi) \quad \text{------(4.2.11)}$$

Where I_0 is bessel function of the first kind, of zero order and with imaginary argument.

Bartlett (1975, 1960) has given a detailed discussion of some data of Gause (1938) on the growth of Paramecium aurelia when feeding on the yeast cells. It is doubtful whether any real ecological situation is sufficiently homogeneous for the simple kind of mathematical model we are discussing to be at all adequate. But, for what it was worth, the data suggested the value $\lambda_1 = 1, \mu_2 \sim 0.45$ (with the time measured in days) and equlibrium point at about (1.5×10^7, 100). Thus $\mu_1 \sim 0.01$, $\lambda_1 \sim 3 \times 10^{-8}$. This gives the period of the cycle as 9.4 days. In Gause's data $\gamma \sim \frac{5}{6} f_2$. Hence $J \sim 2.6$. A typical value of P' was 15. Substitution of these quantities in (4.2.9) gives the chance of extinction at the critical phase of cycle as order $e^{-5.7} \sim 0.003$. Bartlett's discussion

is also supported by simulation studies based on Monte Carlo method. Although all those arguments are very tentative and heruristic in nature, they do provide some qualitative basis for an approximate evaluation of the stochastic aspects.

There is little difficulty in writing down the Partial differential equation for the moment generating function appropriate a fully stochastic model of the Prey-predator to situation. Let the sizes of the species are represented at time t by the random variable $X_1(t)$ and $X_2(t)$. The chance of birth in the first population in time Δt is $\lambda_1 x_1 \Delta t$ and chance of at death is $\mu_1 x_1 x_2 \Delta t$.

The corresponding quantities for the second population or $\lambda_2 x_1 x_2 \Delta t$ and $\mu_2 x_1 \Delta t$. The usual function $f_{1,0} = \lambda_1 x_1, f_{-1,0} = \mu_1 x_1 x_2$, $f_{0,1} = \lambda_2 x_1 x_2$ and $f_{0,-1} = \mu_2 x_2$. Equation established in section 10.9, Bailey, Stochastic Process, gives:

$$\frac{\partial M}{\partial t} = \lambda_1 (e^{\theta_1} - 1) \frac{\partial M}{\partial \theta_1} + \mu_1 (e^{-\theta_1} - 1) \frac{\partial^2 M}{\partial \theta_1 \partial \theta_2} + \lambda_2 (e^{\theta_2} - 1) \frac{\partial^2 M}{\partial \theta_1 \partial \theta_2} + \mu_2 (e^{-\theta_2} - 1) \frac{\partial M}{\partial \theta_2} \quad \text{---(4.2.12)}$$

Equating coefficients of θ_1 amd θ_2 on both sides gives

$$\left.\begin{aligned} \frac{dm_{10}}{dt} &= \lambda_1 m_{10} - \mu_1 m_{11} \\ \frac{dm_{21}}{dt} &= \lambda_2 m_{11} - \mu_2 m_{01} \end{aligned}\right\} \quad \text{---(4.2.13)}$$

which although slightly simpler, still encounters the difficulty of involving two equation with three unknowns which can be solved mathematically.

4.3 AGE-DEPENDENT PREDATION MODEL

The stability of models of age-dependent predation in continuous time with predators exhibiting a functional response are analyzed here. The study of predator prey- systems began with the early work of Lotka and Volterra who treated the simplest cases. In recent years to understand better the dynamical behaviour of predator-prey systems various complications have included (see, e.g., the review, Murdoch and Oaten, 1975). One complication that is certainly present is that predators do not eat all ages or sizes of prey indiscriminately. Numerous examples ranging from mollusks to insects to fish illustrate this point.

Some of the best documented cases of age-dependent predation are from fish. Nielsen (1980) discusses the interaction between walleye and yellow perch, where the major diet item for adult walleye is juvenile yellow perch (see also Le Cren et al. 1977, and references in these papers). Paine (1965) discusses how there is a maximum size to prey consumed by Navanax inermis, an opisthobranch. Dayton (1971) discusses how Balanus cariosus and Mytilus californianus escape predation by Thais by growing to a large enough size. Many insects are preyed upon only as adults. A spectacular example of this is the periodical cicada (Lloyd and Dybas, 1966) Age dependent predation is also important in cases where the prey is an ungulate as in the interaction between mouse and wolves on Isle Royale (Jordan et al., 1971). A simple model for this case was analyzed by Gazis et al (1973).

Predation only on Jurenilies or only on adults has been treated in a number of continuous-time models in addition to those mentioned above. For the simple interaction terms used by Lotka and Volterra, predation on only juveniles or only on adults has been shown to act as a stabilizing influence. In fact, the model, which is originally neutrally stable, becomes stable for all values of the parameters (May, 1974; Smith and Mead, 1974). Similar results obtain for the studies mentioned earlier. Another model, where the predations eats only "eggs" seems to indicate that age-dependent predation cannot be stabilizing, and in fact may be destabilizing (Gurtin and Levine, 1979).

In fact, age-dependent predation has been extensively discussed in the context of "prudent predation" (Slobodkin, 1974). This discussion centered on the evolution of prodent predation, partly as a means of leading to stable predator-prey systems. Missing was a detailed account of the stability properties of dynamical models with age-dependent predation. A particular set of models was analyzed by Maynard Smith and Slatkin (1973).

In the current Section, the age-dependent predation in continuous time models shall be studied and also an arbitrary functional response by the predator shall be included. Since many if not most reasonable functional responses are destabilizing (Murdoch and Oaten, 1975), the question of whether age dependent predation can overcome the destabilizing effect and lead to a model with a stable equilibrium is important. The answers turn out to be more complex and interesting than at first supposed.

More specific questions than whether age-dependent predation is stabilizing are also important. One question is whether predation only on juveniles or only on adults is in some sense more stabilizing. Another question is whether the models always become more stable as the proportion of the population at risk declines (or the proportion safe from predation increases), Other questions will arise in the course of the analysis.

THE MODELS

The form of the investigation here will be to examine a series of models and compare the outcomes from each. A single truly general model would be extremely complex, so it resorts to consider a number of specific models. A series of models is necessary to examine the robustness of the results obtained. The results will be presented in the following section. The various models considered are listed in Table 1.

The set of models for the case where predation is only on adults shall first be described.

TABLE-1

Models Studied and the Equations in Which they Are Defined:

	Predation		
	Only on adults	Only on Juveniles	On all stages
Fixed Juvenile period	(4)	(13)	
Two variable juveile stages	(6)	(15)	
One variable juvenile stage	(5)	(14)	(7)

Let $H(t)$ denote the number of adult prey at time t, and let $P(t)$ denote the number of predators at time t. In the absence of predation, the prey will be assumed to obey the following linear equation, which allows for a juvenile period:

$$\frac{dH}{dt} = r\int_0^\infty H(t-s)\,G(s)\,ds - DH \qquad \text{.....(4.3.1)}$$

where $G(z)$ is the probability that an individual

survives to age z and matures from Juvenile to adult at age z,r is the per capita birth rate, and D is the death rate of adults.

If there is predator with a functional response, the equation for the prey dynamics becomes

$$\frac{dH}{dt} = r\int_0^\infty H(t-s)\,G(s)\,ds - DH - Pf(H) \qquad \text{---(4.3.2)}$$

where f(H) is the functional response of predators to prey (see, e.g., Murdoch and Oaten, 1975).

The predator population will obey the equation

$$dP/dt = cPf(H) - kP, \qquad \text{------(4.3.3)}$$

where c represents the conversion rate of prey deaths into predator births and k is the death rate for the predator population.

The specification of the model will be complete once the maturity function G(s) and the functional response f(H) are specified. Now, leave the functional response general, since only its derivative enters into the determination of stability. In fact, for the qualitative results here what is most important is whether the response is stabilizing or destabilizing as in the work of Murdoch and Oaten (1975).

For the function G(z) one must be more specific. Now, choose several specific forms, including those distributions which allows one to convert the problem to an equivalent one with extra ordinary differential equations (McDonald, 1978. pp.13-21). Concentrate on two extreme cases, namely, those leading to the following two models, which are special cases of the model

described by Eqs.(4.[illegible]) and (4.[illegible]). The cases correspond to two forms for G(z) that give no variance in the length of the juvenile period or very large variance. The amount of variance in the juvenile period will be extremely important in the behaviour of the models.

PREDATION ON ADULTS

Fixed juvenile Period:- The models can be more easily understood by direct derivation, however. The first one says that the prey has a juvenile period of fixed length T. Only adults are subject to predation, and only adults can reproduce. This leads to the model

$$dH/dt = rH(t-T) - DH - Pf(H), \quad \text{.....} (4.3.4a)$$
$$dP/dt = cPf(H) - kP \quad \text{.....} (4.3.4b)$$

One Variable Juvenile Stage:-
The other possibility that can be considered in detail is the case where there is a maturity rate from juvenile to adult, instead of a fixed period. This leads to the model

$$dH_o/dt = rH_1 - d_oH_o - aH_o \quad \text{.....} (4.3.5a)$$
$$dH_1/dt = aH_o - d_1H_1 - Pf(H_1) \quad \text{.....} (4.3.5b)$$
$$dP/dt = cPf(H_1) - kP \quad \text{.....} (4.3.5c)$$

The variables H_o and H_1 are the sizes of the Juvenile prey population and the adult prey population, respectively. The new symbols d_o and d_1 represent predator-independent death rates of Juveniles and adults, respectively. The maturity rate is a, which determines the mean length of the juvenile period.

Two Variable Juvenile Stages: As a link

between the two models above. consider the following model, in which there are two juvenile stages. In fact, the model with a fixed juvenile period can be thought of as one with an infinite number of juvenile stages (McDonald, 1978, p. 15). The model with two stages is

$$dH_0/dt = rH_2 - d_oH_o - aH_o, \quad \text{---- (4.3.6a)}$$

$$dH_1/dt = aH_o - d_1H_2 - aH_1, \quad \text{---(4.3.6b)}$$
$$dH_2/dt = aH_1 - d_2H_2 - Pf(H_2), \quad \text{---(4.3.6c)}$$
$$dP/dt = cPf(H_2) - kP \quad \text{---- (4.3.6d)}$$

The meaning of the new symbols is analogous to the uses in the previous models, where H_2 is now the (number of the) adult stage.

Predation on all Stages — One Variable Juvenile Stage

Before proceeding to the models which include predation only on juveniles, [illegible] introduce the following model, which is the analog of model (4.3.5) with one juvenile stage, but allowing predation on all stage. This serves as a comparison for the stabilizing influence of age dependent predation. The model is

$$dH_o/dt = rH_1 - d_oH_o - aH_o - Pf(H_o + H_1)H_o/(H_o + H_1), \quad \text{----(4.3.7a)}$$
$$dH_1/dt = aH_o - d_1H_1 - Pf(H_o+H_1)H_1/(H_o+H_1) \quad \text{---(4.3.7b)}$$

$$dP/dt = cpf(H_o+H_1) - kP \quad \text{-- (4.3.7c)}$$

PREDATION ONLY ON JUVENILES

The first model with predation only on juveniles that can be described is the analog

of model (4.3.7), with a fixed juvenile period. Let $h_j(a,t)$ be a denisty function on age a, at time t, for the number of juvenile prey. Let T be the length of the Juvenile period. Then the total number of juveniles is

$$H_j(t) = \int_0^T h_j(a,t))da. \qquad \text{-----(4.3.8)}$$

Let $H_a(t)$ be the number of adult prey at time t. Then the adult prey population will obey the equation

$$dH_a/dt = h_j(a,T) - dH \qquad \text{-----(4.3.9)}$$

where d is the death rate of adults. The juvenile population will obey a von Foerster-type equation, namely;

$$\partial h_j/\partial t + \partial h_j/\partial a = -f(H_j)P, \qquad \text{-----(4.3.10)}$$

where the only cause of death in juveniles is predation. Additionally, (4.3.10) has a boundary condition representing births, namely,

$$h_j(0,t) = bH_a \qquad \text{-----(4.3.11)}$$

The predator population will obey the equation

$$dP/dt = PH_j - kP \qquad \text{-----(4.3.12)}$$

Fixed Juvenile Period:- The model can be simplified somewhat by solving the partial differential equation (4.3.7), using boundary condition (4.3.8), assuming $f(H_j) = H_j$, and substituting in

and yielding

$$dH_a/dt = bH_a(t-T)\exp\left\{-\int_0^T P(t-s)\right\} - dH_a \quad \cdots (4.5.13a)$$

$$dP/dt = P\int_0^T bH_a(t-a)\exp\left\{-\int_0^a P(t-s)\,ds\right\}da - kP \quad \cdots (4.3.13b)$$

Unfortunately, the model represented by (4.) is still quite unwieldy, and will not be analyzed here. Clues to its almost certain curious behaviour are to be found below, however.

One Variable Juvenile State:- Models will now be developed to illustrate the effects of predation only on juveniles that can be analyzed, at least in part. There are the analogs of models and with juvenile stages. Hence consider the model

$$dH_o/dt = rH_1 - d_oH_o - aH_o - Pf(H_o), \quad \cdots (4.3.14a)$$
$$dH_1/dt = aH_o - d_1H_1 \quad \cdots (4.3.14b)$$
$$dP/dt = cPf(H_o) - kP \quad \cdots (4.3.14c)$$

TWO VARIABLE JUVENILE STATES:- The appropriate analog to model ( ) is the following one, where predation includes both juveniles stages, since it is the extension of this model that would eventually lead to the model embodied in ( ) and ( ). Hence, consider the model

$$dH_o/dt = rH_2 - d_oH_o - aH_o - Pf_o^*(H_o), \quad \cdots (4.3.15a)$$
$$dH_1/dt = aH_o - d_1H_1 - aH_1 - Pf_1^*(H_1), \quad \cdots (4.3.15b)$$
$$dH_2/dt = aH_1 - d_2H_2, \quad \cdots (4.3.15c)$$
$$dP/dt = cPf(H_o+H_1) - kP, \quad \cdots (4.3.15d)$$

where

$$f_i^*(H_i) = f(H_0+H_1)H_i/(H_0+H_i). \qquad (4.7.16)$$

Although this model will prove too difficult to analyze in great detail, some of its exciting behaviour will become apparent in special cases.

The results are in the form of a stability analysis of the unique nontrivial equilibrium point for all the models (when the equilibrium exists), supplemented by numerical integration. All calculations are postponed to the Appendix.

In all the models the condition for the existence of the unique nontrivial equilibrium is the same: that the prey population grows (without bound) in the absence of the predator. This can easily be translated into an appropriate algebraic condition for all of the models. Another feature common to all the models is that the equilibrium level of the prey stage at risk does not depend on the length of the juvenile period. This is a consequence of the assumption of no interference among the predators. Only the equilibrium level of the group not at risk changes as the length of the juvenile period changes. In all cases the equilibrium behaviour is simpler when the prey class at risk from predation has no other source of mortality. In this case the survival of the prey in the absence of the predator is independent of the length of the juvenile period.

PREDATION ONLY ON ADULTS

Fixed Juvenile Period:- Model (4.3.4.) with a fixed juvenile period has the following behaviour,

as can be shown using recent results of Cooke and Grossman (1982). If the functional response is stabilizing, then the equilibrium is stable for all values of all the parameters when it exists. It is for the destabilizing functional response that the curious behaviour is present. Then when the juvenile period T = 0, the nontrivial equilibrium is unstable. As T increases the equilibrium may become stable, then unstable, stable, etc., with any number of switches possible. (This is illustrated for some examples in Table 2.) For sufficiently large values of T the equilibrium is unstable. The results can be understood as an interplay between a resonance effect and the destabilizing effect of time delays. The time delays for which the model is unstable represent the length of the predator-prey oscillation (see Table 2). Hence the first set of switches is biologically most meaningful. Note that depending on the length of the juvenile period, either an increase or a decrease in the juvenile period can be either stabilizing or destabilizing.

Another consequence of the switching behaviour is apparent from Table 2. Either an increase or a decrease in the other parameters of the model, namely the birth rate b, and adult death rate d, can be either stabilizing or destabilizing. In a similar fashion either an increase or a decrease in the death rate of the predator can be stabilizing or destabilizing.

For almost all parameter values except those with every large values of r/k(prey birth rate to predator death rate) or extremely destabilizing functional responses, there is at

least one interval of stability (in terms of the length of *

TABLE - 2

Stability Results for Model 4.3.1 with Fixed Juvenile Period and Predation Only on Adults

r	D	q	n	$\tau_{n,2}$	$\tau_{n,1}$	$(n+1)$ period
0.04	0.02	0.8	0	3.82	43.04	49.68
			1	57.03	89.41	99.36
			2	110.25	135.77	149.04
			3	163.46	182.14	198.72
			4	216.68	228.51	248.40
			5	269.89	274.88	298.08
0.2	0.1	0.8	0	1.86	17.69	22.23
			1	27.76	36.74	44.46
			2	53.66	55.79	66.68
2.0	1.0	0.8	0	0.81	4.08	7.07
10.0	6.0	0.8	0	0.58	1.27	3.60
0.4	0.1	0.5	0	3.43	9.39	16.54

The equilibrium point is stable only for T values (juvenile periods) lying between $\tau_{n,2}$ and $\tau_{n,1}$ as shown. The last column is multiples of the period of the predator-prey oscillation with no juvenile period, as determined by linearization.

*the juvenile period.) Multiple switches require small values of r/K or only slightly destabilizing functional responses. Here small values of r/k are those in the range 0-0.1, which would represent very long lived prey. Large values would be greater than 10 representing very short lived prey. A very destabilizing functional response would have a slope significantly less than half that of the Lotka-Volterra (linear) response at the equilibrium.

Numerical integration of this model indicates that the trajectories get very close to the axes for those parameter values for which the equilibrium is unstable. Hence, the stability of the equilibrium point here may be a good indication of the possibility for persistence of the system.

One Variable Juvenile Stage:- The behaviour of the model with a single juvenile period is simple by comparison. Only one switch from instability to stability is possible. If the functional response is stabilizing stable for all parameter values. If the functional response is destabilizing then stable if and only if the maturation rate is slow enough.

Two Variable Juvenile Stages:- Not suprisingly, the model with two juvenile stages is already complex enough to allow several switches. This is expected, since model 4.34 can be represented as one with an infinite number of juvenile stages. The variance in the juvenile period is already small enough in this case for the resonance effect to occur.

Predation on All Stages - One Variable Juvenile Stage :

The stability of the equilibrium of model 4.3.7 with two prey stages and age independent predation provides a standard. Here stability is determined solely by whether the functional response is stabilizing or destabilizing.

Predation Only on Juveniles

One Variable Juvenile Stage. The behaviour of the models with predation only on juveniles is of similar complexity, but with some important differences. Now, first consider the model with one juvenile stage. Again, if the functional response is stabilizing, the model is stable independent of the length of the juvenile period. If, however, the functional response is destabilizing, there are two possibilities. The first is that the model has a stable and the second is that the nontrivial equilibrium only if the juvenile period is short enough. The other possibility occurs when the functional response is extremely destabilizing. Then, the model is unstable no matter how short the juvenile period is. The reason for this difference will be discussed below.

Two Variable Juvenile Stages: The model with predation on juveniles with two stages, which represents a reproduction in the variance in the length of the juvenile period, already has very complex behavior. This is apparent even when the functional response is of the simplest form, namely, the linear Lotka-Volterra response. Even this model is too complex to study in detail,

Now we can give several particular cases illustrating the possibilities. First assume that the only cause of death in the juvenile stages is predation, so $d_o=d_1 = 0$). Next, assume (possibility after suitable normalizations) that $c = k = 1$. Finally, in both cases the death rate of the adult prey will be small, with $d_2=0.1$.

(1) Then, if $r = 1$, the model is stable only if the juvenile stages are short enough.

(2) However, if $r = 3.5$, the model is stable only if the juvenile stages are neither too short nor too long. In particular, the necessary and sufficient condition for stability is approximately

$$0.005 < a < 0.02 \quad \cdots\cdots \quad (4.3.17)$$

(3) Still more complicated behaviour is possible. If $r = 2.5$, then the equilibrium is stable if the juvenile period is extremely short, or of intermediate length, with a region of instability in between. The necessary and sufficient condition for stability is approximately

$$0.291 < a \quad \cdots\cdots (4.3.18a)$$

or

$$0.006 < a < 0.061 \quad \cdots\cdots (4.3.18b)$$

Two other possible behaviours are clearly possible in models with more complex functional responses, namely, stability for all lengths of juvenile period, or instability for all lengths of juvenile period.

This last example illustrates another way in which predation only on juvenile is less stable

than predation only on adults. With predation only on adults, and a linear (Lotka - Volterra) functional response the model is stable. With predation only on juveniles, and a linear functional response, the nontrivial equilibrium can be either stable or unstable, depending on the parameters. This confirms in part the results of Gurtin and Levine (1979) for a very different model, but suggests a more complex situation. In their model, predation only on eggs always destabilized a Lotka - Volterra model. For the model here, the results are dependent on the length of the juvenile period and the other parameters of the model.

4.4. ABSOLUTE STABILITY IN PREDATOR PREY MODELS

An equilibrium of a time-lagged population model is said to the absolutely stable if it remains locally stable regardless of the length of the time delay and the criteria for absolute stablity provide a valuable guide to the behavior of population models. For example, it is sometimes assumed that time delays have a limited impact until they exceed the natural time scale of a system; here it is stressed that under some conditions very short time delays can have a marked (and often maximal)destabilizing effect.

Levins (1966) divided population models into three categories depending upon the balance of generality, realism, and precision used in their development. Each type of model has a specific role; those which sacrifice generality to realism and precision are appropriate for the detailed simulation of specific systems; those which sacrifice realism to generality and precision (generally by using linear functions) are appropriate for the initial development of hypotheses; and those which sacrifice precision to realism and generality

are necessary to test these hypotheses and provide the basis for a sound theoretical framework. Precision is lost in this final category by the inclusion of highly nonlinear functions, often specified only by their shape. Analyses of these models usually center upon determining the conditions necessary for the local stability of an equilibrium, and serve to identify those biological factors which tend to promote the stability of systems and those which tend to oppose it (for reviews see May, 1973a; Murdoch and Oaten, 1975; Hassell, 1978).

Even in the final category it is necessary to foresake some aspects of realism and one of the features of the real world which is commonly compromised in order to achieve generality is the time delay. General population models are usually expressed either as undelayed differential equations, in which case the time delays inherent in all biological processses are ignored, or as difference equations, in which case a synchronized time delay of one generation dominates and dynamic features with more immediate action are sacrificed. But do these simplifications affect the results we derive in any important qualitative way? To test the possibility a number of delay-differential equations have been analysed (for reviews see Adams et al, 1980. Nisbet and Gurney, 1982). The results have provided broad support for the thesis that time delays consistently reduce stability, with delays less than the natural time scale of a system (short delays) tending to have little effect, and with delays greater than the natural time scale of a system (long delays) tending to create instability (May, 1973a, b;Maynard Smith, 1974). Further support for this view come from

the analysis of difference equations (see May et al, 1974). However, on occasion contrary effects have been apparent (see, for example, Cushing, 1977, 1980), suggesting that there may be more to the story.

Recent analysis of delay-differential equations have focussed upon the problem of incorporating lags arising from maturation time. Maynard Smith (1974) explicity included a maturation time delay in a single trophic level model and Blythe et al, (1982, 1984) have increased the generality of the approach, while Gurney et al, (1980) have demonstrated that specific models can provide an excellent description of experimental data. An early attempt at incorporating a maturation time delay in a two trophic level predator-prey model was made by Wangersky and Cunningham (1957). Goel et al.,(1971) printed to problems in the analysis but their own solution can be shown to be incomplete; however, Cushing (1981), Cushing and Saleem (1982), Hastings (1983) and Nunney (1983 1985a,b) have now analysed realistic predator-prey models incorporating maturation time

Taken as a whole, this recent work overturns two rules which in the past formed the basis for our view of time delays; that sufficiently long delays create instability, but that short delays have little effect. The lack of generality of these rules suggests the need for a new general framework which can incorporate recent results. To this end Nunney (1983) has stressed the value of examining the criteria for absolute stability. Absolute stability, or more correctly absolute asymptotic stability, is defined as local stability

which perserveres in the presence of a time delay of any length (El'sgol'ts and Norkin, 1973) and the concept is of particular value because it provides criteria which are rebust to changes in the details of a time delay. This insensitivity to detail raises the possibility of understanding in a very general way how time delays interact with the various features of population models.

With this aim in mind, the analysis of a general predator-prey model is presented incorporating maturation time delays due to the predator, the prey, or both. It is shown that in all cases examined conditions guaranteeing absolute stability exist and that qualitatively these conditions fellow a very simple pattern

A PREDATOR-PREY MODEL

Before incorporating time delays into the analysis, it is necessary to define a general predator-prey modelNunney (1985a) introduced the model

$$\left.\begin{aligned} \frac{dP}{dt} &= PF(H) - PM(H) \\ \frac{dH}{dt} &= B(H) - D(H) - PG_1(H) \end{aligned}\right\} \quad \ldots(4.4.1)$$

where P and H define the predator and prey (resource) levels. The per capita birth and death rates of the predator population are functions of food level (predator interference is not included) and are defined by the increasing function F and the decreasing function M, respectively. The birth and death rates of the prey are defined at the population level by the functions B and D.D is an increasing function, whereas B, the prey recruitment curve, may be increasing

(unregulated birth) or decreasing (regulated birth) depending upon where the prey equilibrium lies in relation to the peak of the recruitment curve. Finally the capture rate or functional response of each predator is defined by G(H).

Using the undelayed model 4.4.1, it can easily be shown that an equilibrium will be locally stable if and only if

$$\frac{dD}{dH}+P\frac{dG}{dH}-\frac{dB}{dH}>0 \qquad (4.4.2)$$

This condition, like all other stability conditions given in this Section evaluated at the equilibrium. The potential importance of prey self-regulation ($dB/dH < 0$) and of the accelerating part of the type 3 functional response ($dG/dH > G/H$) in promoting the stability of predator-prey models generalized by (4.4.1) is well known (for a review see Murdoch and Oafen, 1975); however, in time-lagged models an important difference between these two factors must be borne in mind: prey self-regulation may act as a delayed term but the effect of predation is always immediate.

The necessary and sufficient condition for local stability, defined by (4.4.2) depends only upon components of the prey dynamics. The presence of the predator population is felt only indirectly via its regulation of prey death (PdG/dH). It will be seen that the left-hand side of 4.4.2, which will be referred to as the total prey regulation, continues to play a central role in determining local stability in the presence of time delays, but a delay in the predator population has the effect of incorporating features of the predator dynamics into the stability criteria. First, however, consider the simplest case, the effect of a single delay due to prey maturation time.

DELAY DUE TO PREY MATURATION

Assuming that the time taken for the prey (or resource) to mature is T, and that this is long compared to the maturation period of the predator then, model 4.4.1 can be rewritten as

$$\left.\begin{aligned}\frac{dP(t)}{dt} &= P(t)[FH(t))-M(H(t))] \\ \frac{dH(t)}{dt} &= B(H(t-T))-D(H(t))-P(t)G(H(t))\end{aligned}\right\} \quad (4.4.3)$$

It is assumed that prey born at time (t-T) mature at time t and enter the adult population. Furthermore because of differences in ecology, size, or other factors, it is assumed that the juvenile prey are not subject to significant predation and do not compete with the adult prey for food.

It has been shown (Nunney, 1985a) that the model (4.4.3) will retain local stability regardless of the length of the time delay. T, provided that (at the equilibrium)

$$\frac{dD}{dH}+P\frac{dG}{dH} > \left|\frac{dB}{dR}\right| \quad \text{.....} \quad (4.4.4)$$

Hence the inequality (4.4.4) defines the necessary and sufficient condition for adsolute stability. This condition can be viewed as two distinct criteria: First the total prey regulation (dD/dH+PdG/dH-dB/dH) must be positve, as is required in the undelayed system [see 4.4.2]. This condition constrains dB|dH from being too positive. Second, a new feature, the effective prey regulation (dD/dH+PdG/dH+dB/dH), must also be positive. The effective prey regulation treats the delayed effect as potentially destabilizing. Strong regulation of recruitment, when subject to certain time delays, can produce an entirely inappropriate nonregulatory response.

Hence this condition constrains dB/dH from being too negative. It also illustrates an important rule: when viewed over a range of potential delays, it is the magnitude rather than the sign of a delayed process which determines stability.

In an undelayed system, a large negative value of dB/dH reflects strong self-regulation and has a powerful stabilizing effect. Self-regulation at the lowest tropic level is often employed to stabilize model systems (see, for example. Pimm, 1982). The condition(4.4.4) suggests that the stabilizing role of self-regulation via recruitment may be limited. This limitation would be trivial if only very long delays minimized the role of self-regulation (since few organisms could have such an exceedingly long maturation time); however, the beneficial effect of prey self-regulation is lost very rapidly and time lags much less than the natural period of the prodator-prey system can cause a significant loss of stability. This effect can be seen by representing a system with self-regulation acting through recruitment ($dB/dH < 0$). The regulation is strong enough to put the system at the limit of absolute stability. By contrast, the example shown in Figure 1 is at the

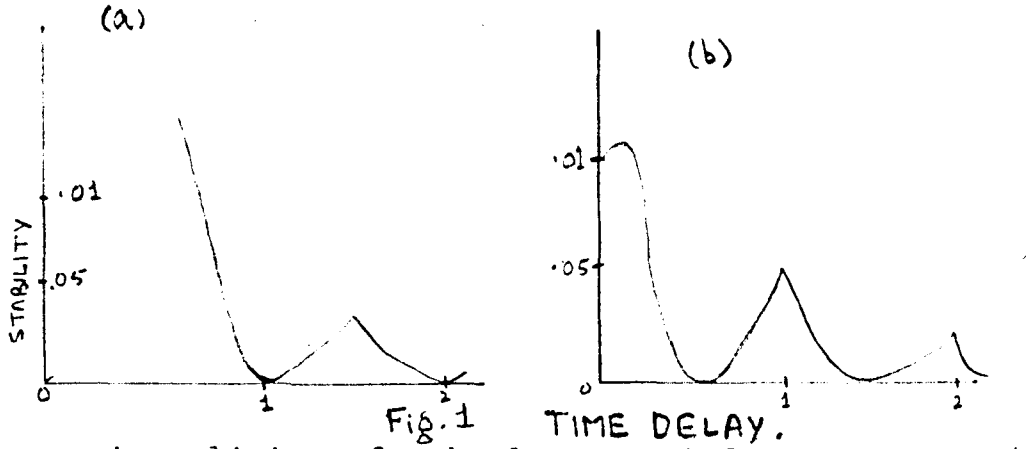
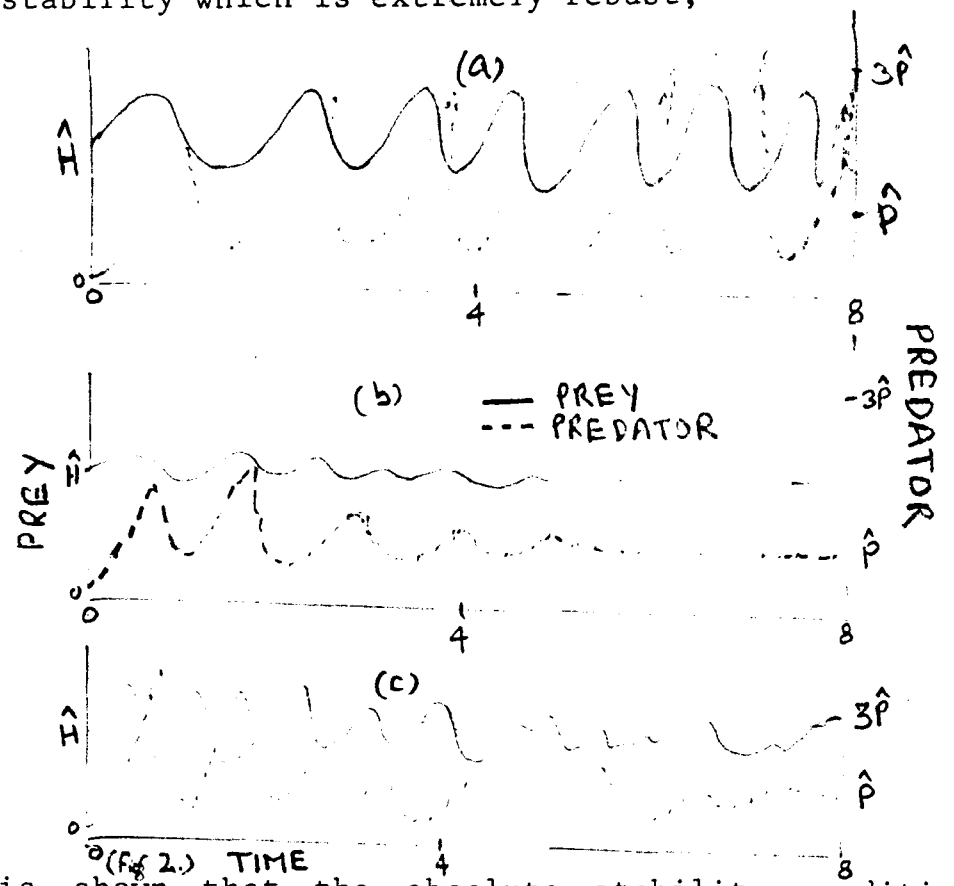


Fig. 1

opposite limit of absolute stability with $dB/dH > 0$. The regulation of prey recruitment is insufficient to stabilize the undelayed system which, like the

Lotka-Volterra model, is neutrally stable. However, given a short time delay in recruitment, the initially neutral equilibrium becomes strongly stabilized. The same effect can also stabilize an initially unstable system (Hastings, 1983). This somewhat unexpected role of a time delay increasing stability is discussed in detail elsewhere (Nunney, 1985b).

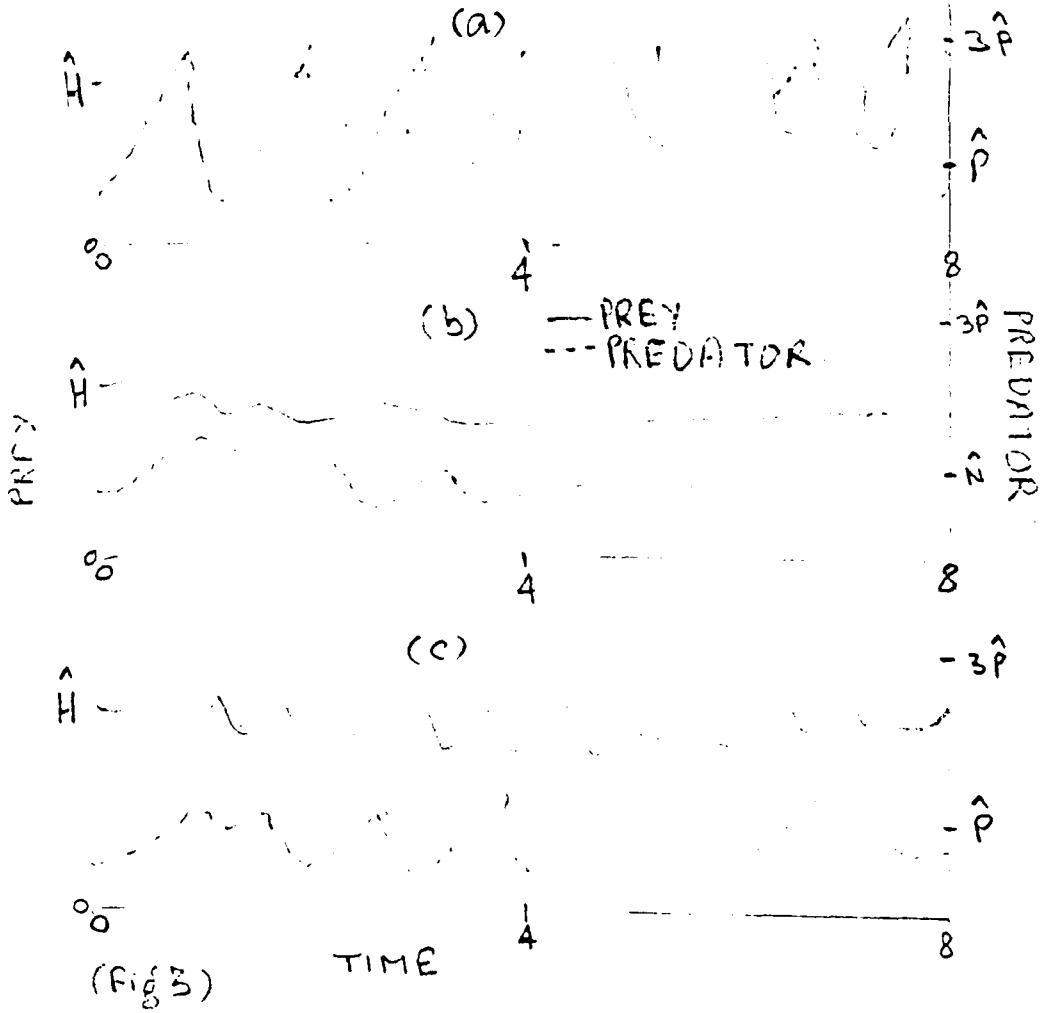
The criterion of absolute stability is not appropriate for examining the details of the interaction between a time delay and stability. Its value is that it provides a sufficient condition for stability which is extremely rebust,



(Fig 2.)

it is shown that the absolute stability condition (4.4.) applies not only when the delay has some precise value, but it also applies when the delay is distributed, i.e., when different individuals have different maturation times. Furthermore, the same condition generally guarantees stability when the length of the delay changes randomly over time. This occurs because, although the variation causes

fluctuations in the stability of the system (measured by the rate of return towards the equilibrium following a perturbation), the system never develops a tendency to leave the equilibrium. Note that this argument does not apply to variation which is



(Fig 3)

non-random with respect to the system, such as occurs, for example, juvenile prey mature faster when the prey population is low.

To illustrate these points, simulations of the systems used. In each case the system is simulated under three sets of conditions:(a) with the delay which defines the limit to absolute stability (i.e., the most destabilizing delay); (b) with a distributing delay with the same mean as (a); and (c) with a distributed delay with the same mean as (a); and (c) with a variable delay,

which again has the same mean as (a). It can be seen that the systems (b) and (c) are always stable although the parameters define a system at the limit of absolute stability. Not surprisingly the stochastic nature of the variable delay makes (c) less stable than (b) however, it is clear that the criteria of absolute stability provides a valuable conservative indication of stability

DELAY DUE TO PREDATOR MATURATION

If a predator take a time T to mature, and if T is long compared to the maturation time of the prey, when the model(4.4.1)can be rewritten as

$$\left.\begin{aligned}\frac{dP(t)}{dt} &= P(t-T)F(H(t-T))-P(t)M(H(t))\\ \frac{dH(t)}{dt} &= B(H(t))-D(H(t)-P(t)G(H(t)\end{aligned}\right\} \quad \ldots(4.4.5)$$

This form of delay relies upon the implicit assumption that the survival of juvenile predators is not dependent upon the prey level and that these juveniles have no significant impact upon the level of the prey. As in the previous example of a prey delay, this assumption serves to highlight the difference between delayed and undelayed models by preventing the juveniles from having any dynamic role.

Nunner (1985a) has shown that, as T approaches
w nfinity, the system(4.4.5)will be stable if

th $$\frac{dD}{dH}+\frac{PdG}{dH}-\frac{dB}{dH}>\frac{PG}{2M}\left[\frac{dF}{dH}+\frac{dM}{dH}\right] \quad \ldots\ldots\ldots(4.4.6)$$

s
, evaluated at the equilibrium) provided that dM/dH is not too large. We can usefully simplify this form. The left-hand side of4.4.6, the total prey regulation, can be normalised by M. This term

will be defined by P'. Further, we define two terms, also normalized by M, which encapsulate effects acting on the predator population, one derived from the time delayed or "lagged" processes (L) and the other derived from the non-delayed or "immediate" processes (I):

$$L = \frac{PG}{2M}\frac{d(\ln F)}{dH} \quad (\geq 0) \quad \cdots\cdots (4.4.7a)$$

$$I = -\frac{PG}{2M}\frac{d(\ln M)}{dH} \quad (\geq 0) \quad \cdots\cdots (4.4.7b)$$

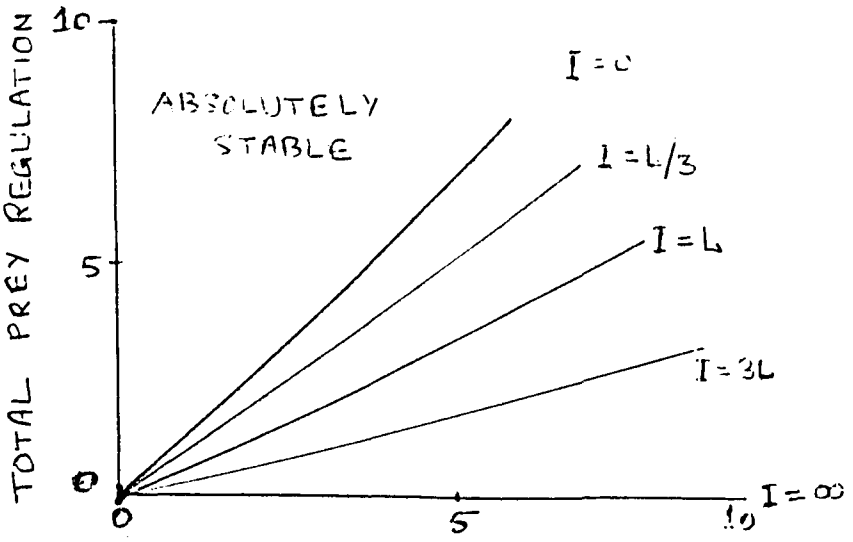
It can be shown that how the condition 4.4.6, which can now be expressed in the form $P' > L - 1$, must be modified when dM/dH becomes too large. This allows as to determine the necessary and sufficient conditions for absolute stability as

$$P' > L - I \quad \text{when} \quad P'^2/4 > I \quad \cdots\cdots (4.4.8a)$$

$$P' > L - I + \frac{(I - P'^2/4)^2}{(L - I)} \quad \text{when} \quad P'^2/4 < I \quad \cdots\cdots (4.4.8b)$$

The conditions (4.4.8) show that the total prey regulation, P^1, which is not directly affected by the delay, continues to promote stability as it would in the undelayed model [e.f. condition 4.4.2]. The delayed term L, acts directly to reduce stability as we might expect. Less expected is the role of the undelayed component of predator dynamics, I, which acts to offset the destabilizing

influence of the time delay. However, the immediate effect, I,



DELAYED PREDATOR EFFECT

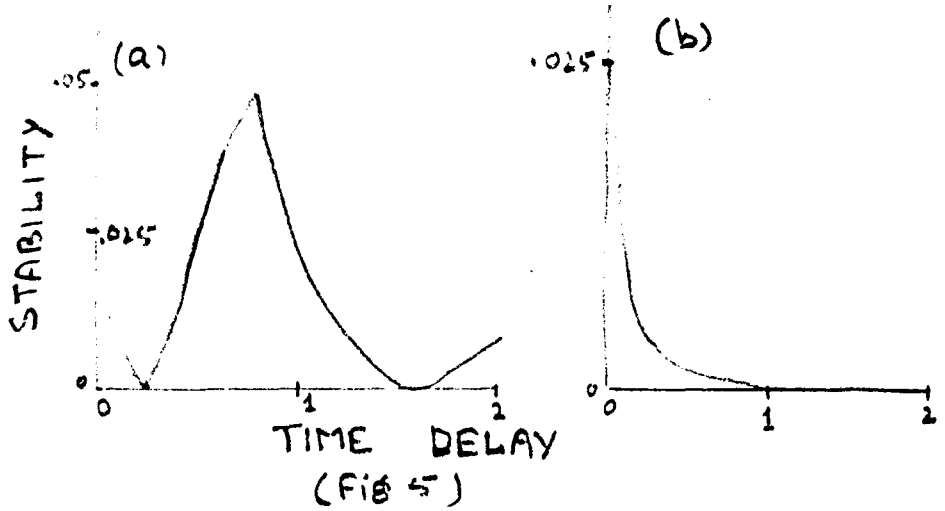
(Fig.4.)

never
can completely offset the lagged effect, L; condition (4.48b) ensures that the total prey regulation, P', must always be positive even when I is very large. This pattern can be shown where the region of absolute stability is illustrated for various proportional relationships between I and L. As I increases relative to L the zone of stability is enlarged: however, even when I is zero a significant zone of stability exists.

It is important to note that in considering the parameter space, changes in the equilibrium values of the predator and prey need not be considered. These values are determined directly by the functions involved in the dynamics, whereas regulation is determined primarily by the first derivative of these functions. There is no prior reason why these two feature should covary and as a result it is possible to treverse all of the parameter space without affecting the equilibrium values.

As in the case of a prey delay, one reason why the conditions for absolute stability are important is that very short delays can dramatically change stability: indeed this effect is more marked for a predator delay than a prey delay. The two examples illustrated differ in the ratio of L to 1 and consequently differ considerably in the shape of their stability curves; however, in both a delay of only one-tenth of the natural period results in a dramatic loss of stability. It can be seen that when I is relatively large, stability does not always decline in the presence of a time delay; the addition of a time delay of around half the natural period makes the system more stable. This type of complex pattern is somewhat similar to that observed by Cushing (1981) and Cushing and Seleem (1982).

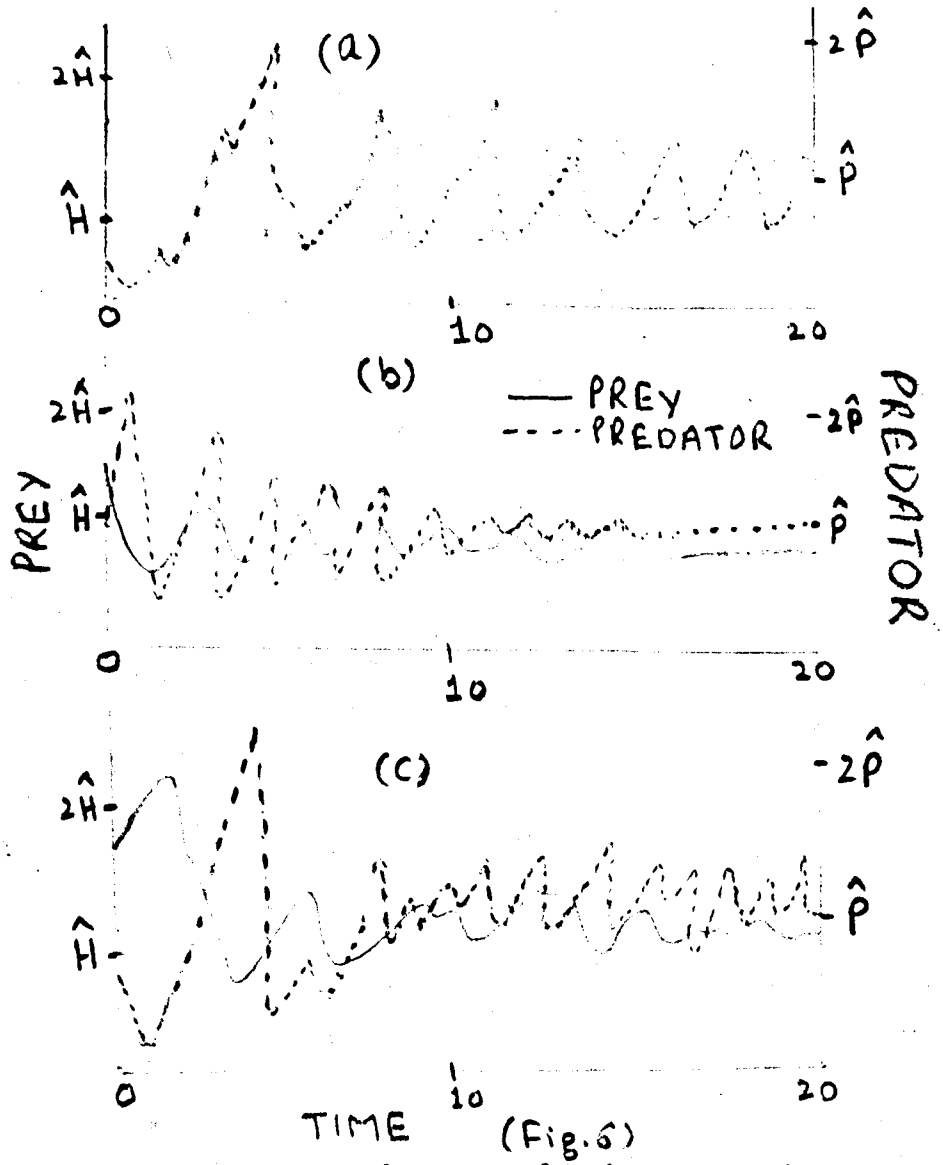
The derivation of the conditions 4.4.3 defining absolute stability in the



(Fig 5)

presence of a predator delay, is complex and for this reason no attempt was made to prove that they quantee stability given any distributed delay, although it is probable that no distributed delay would be more destabilizing than the most disruptive exact delay, relationship can be shown. The relationship is a more extreme example in that the zone around the most destabilizing short delay

defines much greater stability. It has already been shown how variance around the most destabilizing delay enhances stability. However, this



(Fig.6)

argument cannot be applied to the monotonic stability-delay relationship shown in Fig 5b. Instead, the system was simulated using a fixed, distributed or variable delay, and the results are shown in Fig 6. The least stable system is that with a fixed delay, providing further support for the view that the criteria for absolute stability are robust in the presence of distributed or varying delay.

DELAY IN BOTH PREDATOR AND PREY

If both the predators and the prey are subject to a maturation period of T then the model 4.4.1) becomes

$$\left.\begin{aligned}\frac{dP(t)}{dt} &= P(t-T)F(H(t-T)) - P(t)M(H(t)) \\ \frac{dH(t)}{dt} &= B(R(t-T)) - D(H(t)) - P(t)G(H(t))\end{aligned}\right\} \quad \text{---(4.4.9)}$$

As in the two previous delay models, 4.4.3) and 4.4.5), it is assumed that the juveniles have no impact upon the dynamics of the system except via their effect in delaying the response of the populations to changed conditions.

The complete analysis of the absolute stability criteria of 4.4.9) would be very complex. Of particular interest are not the precise stability criteria but whether or not the general principles observed to apply in the presence of a predator delay or a prey delay continue to apply when the two types of delay are combined.

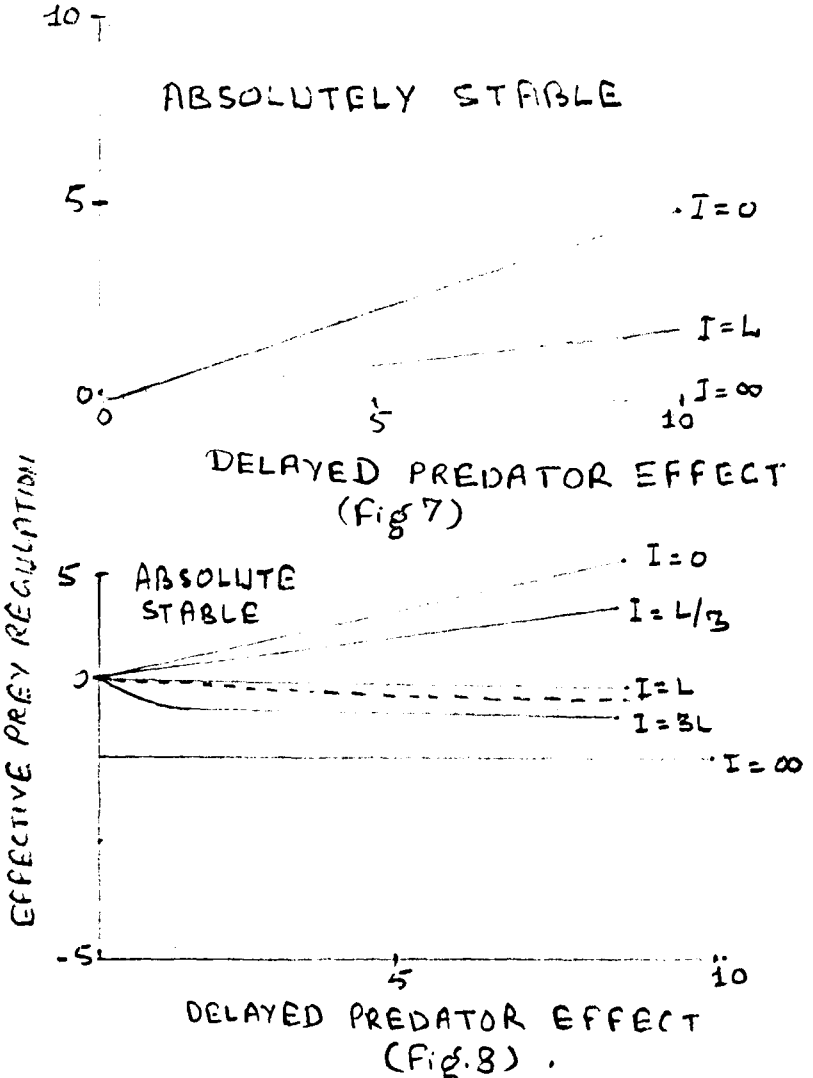
The condition 4.4.4) requires that in the presence of a prey delay both the total prey regulation (dD/dH/+PdG/dH-dB/dH) and the effective prey regulation (dD/dH+PdG/dH+dB/dH) must be positive. The condition 4.4.8) requires that in the presence of a predator delay the total prey regulation must be greater than some positive value. The size of this value

rises as the effect of delayed predator recruitment is increased, althogh this rise can be offset by a similar increase in the effdect of undelayed predator mortality. Combining the general principles of4.4.4) and4.4.8) suggestes that when both the predator and the prey are subject to a delay the total prey regulation will be constrained above zero, but not as severely constrained as would be expected from4.4.8). On the other hand we could expect the effective prey regulation to be more severly constrained, reflecting the destabilizing effects of delayed regulation acting through both prey and predator recruitment.

Before examining these expectations we need to realize that the behaviour of the double-delayed system4.4.9) depends upon the relative importance of the two delays. For example, dB/dH is a measure of the control acting through the prey delay and setting dB/dH=0 removes the dydnamic effect of the prey delay. Similarly, dF/dH is a measure of the control acting through the predator delay although setting dF/dH=0 does not negate the effect of a predator delay: the value of M, which measures the equilibrium per capita birth and death rates of the predators, also acts as a vehicle through which the influence of the delay can be felt. In order to investigate the joint effect of a predator and a prey delay this information to be used and define a system which approximately balances the two delays. To this end the effect of the prey delay is set, as measured by the mangnitude of dB/dH and normalized by M, equal to the delayed predator effect L, which is proportional to dF/dH and also normalised by M (see4.4.7a). The absolute stability of such a system is summarized in Figures. As was the case for the previous models, it is

important to realize that the parameter space shown in these figures can be traversed without alterning the equilibrium values.

A comparison illusrates that the constraints upon the total prey regulation are markedly decreased by adding a prey delay to a system already including a predator delay. In fact, with regard to the total prey regulation, the double-delayed system is hardly more constrained than the undelayed system. Furthermore, this difference becomes



DELAYED PREDATOR EFFECT
(Fig 7)

DELAYED PREDATOR EFFECT
(Fig.8).

vanishingly small as the undelayed part of the predator dynamics, as measured by I (see 4.7b), is increased relating to the lagged part, L.

As predicted earlier the pattern is very different when we look at constraints upon the effective prey regulation. Such constraints reflect the fact that when prey recruitment is subject to a delay, strong regulation of the process can be destabilized. Reference to Figure shows that the addition of a predator delay has a fairly straight-forward effect. When the delayed predator effect (L) is more-or-less equal to the immediate predator effect (I) then the effective prey regulation is constrained as if there was no predator delay. However, if L > I then the constraint can be markedly increased. In fact, as Nunney (1985a) has noted, whenever L is somwhat larger than I then the condition constraining prey regulation of recruitment ($dB/dH < 0$) is

$$\frac{dD}{dH} + P\frac{dG}{dH} + \frac{dB}{dH} > \frac{(L-I)M}{2} \quad \ldots (4.4.10)$$

If L < I then the constraint upon the left-hand side of (4.4.10), the effective prey regulation, is lessened. In fact, a large value of I can offset the effect of the predator delay plus part of the effect of the prey delay so that the constraint can become less than when only the prey are subject to a delay. However, the effect is limited (see Fig 8) because even when I is infinite the effective prey regulation must be greater than - 2M so that under all conditions there is a danger of overly strong regulation of prey recruitment.

DISCUSSION

The analysis of population dynamic models incorporating age structure a complex and, notwithstanding the significant advances that have been made in the recent past (see Cushing, 1980, 1981; Cushing and Saleem, 1982; Gurney et al, 1983), is likely to remain so. One way to approximate the effects of age structure is to add maturation time delays to otherwise continuous models (for example, Maynard Smith, 1974; Gurney et al, 1980; Blythe et al, 1982, 1984). It has been shown that a broad range of such systems can retain stability in the present of arbitrarily long delays (Nunney, 1985a) and that short maturation delays can interact with the natural time scale of a model so that, depending upon the circumstances, the addition of a short delay can result in a marked incresse or a marked decrease in stability (Hastings, 1983; Nunney, 1983, 1985b). Even in a more complex age-structure model it has been shown that decreasing the length of a time delay can cause instability (Cushing, 1981; Cushing and Saleem, 1982.

Such diverse behavior needs to be viewed in some general framework, and here it is argued that such a framework can be developed using the concept of absolute stability. It has been shown that the criteria for absolute stability, which guarantee local stability, regardless of the length of a time delay, are particularly useful in ecological systems because they provide a conservative estimate of stability which is robust in the presence of a distributed delay or in the presence of a delay which tends to very over time. In addition, it has been shown that either a prey or a predator delay can cause maximum instability when short.

This observation, like that of Cushing (1981) and Cushing and Saleem (1982) mentioned above, contrasts with the traditional view that time delays are disruptive only when they are long and makes it necessary to recognize that quite short (and hence biologically realistic) maturation times can have significant dynamic consequences.

In summary, the analysis of the absolute stability criteria of the predator-prey model **(4.4.1)** when subject to various sorts of delay highlights two major points. The first is the repeated theme that the disruptive effect of time-lagged processes can be nautralized by processes acting without delay. The second is that the principles derived from examining the action of a prey delay and of a predator separately can be used to predict the conditions applying when the model is subject to a double delay. It is therefore likely that the general principles derived from the absolute stability criteria of fairly simple models can be applied to much more complex systems. This would be valuable because, given the finding that delays in predator-prey systems can have a significant (and often maximal) disruptive effect when considerably less than the natural period, it appears that only a knowledge of the criteria for absolute stability will allow us to evaluate the dynamic role of various biological phenomena without the nagging doubt that a simple time lag will upset the elegant theory.

4.5 EXTINCTION PROBABILITY IN PREDATOR-PREY MODELS

The predator-prey process was originally studied by Lotka (1925) and Volterra (1926). They considered the deterministic model

$$\left.\begin{aligned}\frac{dP}{dt} &= \alpha PH - \beta P \\ \frac{dH}{dt} &= \lambda H - \mu PH\end{aligned}\right\} \quad \text{.....(4.5.1)}$$

where $P(t) \geq 0$, $H(t) \geq 0$ represent the numbers of predators and prey, respectively, alive at time t. It is well known that the solution to Equations (4.5.1) consists of the set of closed curves

$$\alpha H - \beta \log H + \mu P - \lambda \log P = \text{constant}.$$

round the equilibrium point $(M.N) = (\lambda/\mu, \beta/\alpha)$, and so neither population ever becomes extinct. In this section some properties of stochastic models of predator-prey populations shall be studied: in particular the probability that the predator population eventually becomes extinct shall be investigated.

Relatively little work has been carried out on extinction probabilities using stochastic models. Bartlett (1957) derived an approximation to the small probability that the predators would become extinct during the course of a single cycle round the equilibrium position. Holgate (1976) developed this approximation further and also derived an approximation to the extinction probability by assuming initially that predator behaviour could be considered as a linear birth-death process conditional on the size of the prey population remaining fixed. Barnett (1962) carried out some simulation studies on extinction probabilities for a related ecological model, the competition process.

In a series of papers, Smith and Mead have investigated the extinction of predator-prey models. Smith and Mead (1974) considered three stochastic models, of which one is essentially Model 2 of this Section and the other two include age-dependence. They carried out an extensive simulation experiment, with a range of parameter values, to estimate the probability that the predators become extinct first and the time to extinction of one of the population. In a second Section (Smith and Mead (1979) they applied the approximation technique developed by Bartlett (1957) to their three models and found that the conclusions agreed well with their simulated results as regards the probability of predator extinction, but not on time extinction. They also derived an approximation to the stochastic process using a finite Markovchain (Smith and Mead (1980) and this method was found to give good predications of the time to exinction. In all their work, Smith and Mead assumed that the process was initially at the deterministic equilibrium position.

Other workers have considered a stochastic form of the Lotka, Volterra equations in which the parameters are random variables (see, for example, Gard and Kannan (1976). This approach involves completely different methods and we shall not discuss it further.

Two stochastic models of the predator-prey process are considered here which can be derived from (4.5.1).

MODEL-I:

HOST-PARASITE: In this model it is assumed that parasites (predators P) encounter hosts (prey, H) at random; when such an encounter occurs, one egg is laid which develops into a new parasite and the host dies. Time delays are ignored and we represent this by a Markov process with instantaneous transition rates $q(P,H;\xi,\eta)$ from state (P,H) to state (ξ,η). The only possible events are birth of a host, death of a parasite and parasitism. Hence

$$q(P,H;P,H+1)=\lambda H, \quad q(P,H;P-1,H)=\beta P,$$
$$q(P,H;P+1,H-1)=\alpha PH$$

All other

$q(P,H;\xi,\eta)$ for $(\xi,\eta)\neq(P,H)$ are 0.

Since $\Sigma_{\xi,\eta}\, q(P,H;\xi,\eta)=0$,

$$q(P,H;P,H)=-(\lambda H+\beta P+\alpha PH).$$

This model is a stochastic analogue of (4.5.1) for the special case $\mu=\alpha$.

MODEL 2: This model represents a predator-prey process when a predator birth is not directly associated with a prey death. In this case the transition rates are given by

$$\left.\begin{aligned} &q(P,H;P,H+1)=\lambda H,\ q(P,H;P,H-1)=\mu PH,\\ &q(P,H;P+1,H)=\alpha PH,\ q(P,H;P-1,H)=\beta P,\\ &q(P,H;P,H)=-(\lambda H+\mu PH+\alpha PH+\beta P). \end{aligned}\right\} \quad ---(4.5.2)$$

Becker (1973) considered a similar model in which the prey form only a small proportion of the diet of the predators and hence the predator birth rate is unaffected by the size of the prey population. His model differs from Model 2 only in that $q(P,H,P+1,H) = \alpha P$. When $\lambda = 0$ Becker derived the distribution of the size of the prey population at time t. Billard (1977) considered Model 2 for the special case $\lambda = \alpha = 0$.

CERTAINTY OF ULTIMATE EXTINCTION.

In this section it is shown by using the criteria developed by Reuter (1957), (1961) that ultimate extinction of either predators or prey is certain, and that the mean time of extinction is finite.

In both models, once the process hits either axis it cannot leave it. The remaining single population then follows a linear birth, linear

death or linear birth-death process. However, as we are concerned with the behaviour of the two-dimentional model we shall adopt Reuter's (1961) device of freezing the state (P,O) and (O,H) thus making them absorbing. We thus have a set of absorbing states denoted by A and a set of non-absorbing state (P,H) (where P,H=1,2...) denoted by D.

Reuter (1961) proves that the following result holds (his criterion C): suppose the Markov process with transition matrix Q is regular and has sets of non-absorbing and absorbing states D and A respectively; let $\alpha_i, i \in D$ be the probability of reaching some state in A from i and let $\tau_i, i \in D$, be the expected time to reach A from i; if there exist finite $u_j \geq 0$ such that

$$\sum_j q_{ij} u_j + 1 \leq 0 \qquad i \in D \quad \ldots (4.5.3)$$

then $\alpha_i = 1$ and $\tau \leq u_i < \infty$, that is, absorption in state A in finite time is certain.

A regular Markov process is a conservative process in which there is only one value of Q which satisfies the Kolmogorov forward equations. Both models developed in Section 1 can be shown to be regular using the criterion proved by Reuter (1957) (his Theorem 7): the Markov process defined by Q is regular if, for each $k > 0$, the equations $kz_i = \Sigma_j q_{ij} z_j$, where $0 \leq z_i \leq 1$, have only the trivial solution $z_i = 0$. The regularity of Model 1 can be proved more easily using the specific result (Theorem 1) derived by Reuter (1961) for competition processes where only transitions to

neighbouring states are permitted.

It can be demonstrated that the existence of $u_j \geq 0$ for Model 2. The same form for u_j, but with different constraints on the constants can be used for Model 1. For any point (ξ, η), uniquely denoted by state j, choose

$$u_j = u(\xi,\eta) = h\xi + k\eta + \frac{A(1-r^{\eta})}{1-r} - \frac{B(1-r^{\xi})}{1-r} \quad \xi,\eta > 0 \qquad \ldots\ldots (4.5.4)$$

where h,k,r,A,B are constants, satisfying

$$h>0,\ K>0,\ 0<r<1,\ A(1-r)>B>0 \qquad \ldots\ldots (4.5.5)$$

These constraints ensure that $u_j \geq 0$.

Now for any point (P,H), we shall define

$$v(1) = v(P,H) = -\sum_{\xi,\eta} q(P,H;\xi,\eta)\, u(\xi,\eta).$$

Then using (4.5.2)

$$v(P,H) - \beta P h = H\{P(\mu k - \alpha h) - \lambda K\} - AHr^{H-1}(\mu P - \lambda r) + BPr^{P-1}(\alpha r H - \beta) \qquad \ldots\ldots (4.5.6)$$

If it is now taken that

$$h > \frac{1}{\beta}, \qquad \ldots\ldots (4.5.7).$$

then $\beta P h > 1$, and in order to satisfy (4.5.3) it is required to show that the right-hand side of (4.5.6) is positive. Impose the further restrictions

$$K > \frac{\alpha h}{\mu}, \qquad r < \frac{\mu}{2\lambda} \qquad \ldots\ldots (4.5.8)$$

let

$$N = \max\left\{ \frac{2\beta}{\alpha r}, \frac{\lambda K}{\mu K - \alpha h} \right\} \quad \ldots (4.5.9)$$

and consider the four regions in the positive quadrant bounded by P= N,H=N separately.

(i) P > N, H > N. In this region,

$$P(\mu K - \alpha h) - \lambda K > N(\mu K - \alpha h) - \lambda K > 0 \quad \text{by (4.5.9)}$$

$$\mu P - \lambda K > \mu P - \frac{\mu}{2} \quad \text{by (4.5.8)}$$

$$> 0;$$

$$\alpha r H - \beta > \alpha r N - \beta$$

$$> \beta \quad \text{by (4.5.9)}$$

$$> 0$$

Hence $v(P,H) > 1$ in this region.

(ii) $P \leq N$, $H > N$. In this region,

$$\alpha r H - \beta > \frac{1}{2}\alpha r H + \frac{1}{2}\alpha r H - \beta$$

$$> \frac{1}{2}\alpha r H \quad \text{by (4.5.9)}$$

Hence

$$B P r^{P-1}(\alpha r H - \beta) > \frac{1}{2} B \alpha H r^N$$

$$> \lambda K H$$

provided we choose $B > 2\lambda k/\alpha r^N \quad \ldots (4.5.10)$

Since $\mu P - \lambda r > 0$ and $\mu k - \alpha h > 0$, the right-hand side of (4.5.6) is positive in this region.

(iii) $P > N, H \leq N$. In this region, $P(\mu k - \alpha h) - \lambda k > 0$ as in (i) and $\mu P - \lambda r > \mu(P-1)$ by (4.5.8). Hence

$$A H r^{H-1}(\mu P - \lambda r) - B P \beta r^{P-1} > A H r^{N-1} \mu P \frac{N-1}{N} - B P \beta r^{N-1} = P r^{N-1}\left(A H \mu \frac{N-1}{N} - B\beta\right)$$

> 0 provided $A > N B \beta / \mu (N-1)$.

(iv) $P \leq N$, $H \leq N$. In this region $\mu P - \lambda r > \mu/2$ by (4.5.8). Hence

$$AHr^{H-1}(\mu P - \lambda r) - BPr^{P-1}\beta - H\lambda K > AHr^{N-1}\frac{\mu}{2} - B\beta N - \lambda KN$$

$$> 0 \text{ provided } A > \frac{2N(B\beta + \lambda k)}{\mu r^{N-1}}$$

So Reuter's criterion, $\Sigma q_{ij} u_j + 1 \leq 0$, is satisfied for all $i \in D$, by taking u_j, given by (4.5.4) where the constants h,k,r,A,B satisfy (4.5.5), (4.5.7), (4.5.8), (4.5.9), (4.5.10) and

$$A > \max\left\{ \frac{B}{1-r} , \frac{2N(B\beta + \lambda K)}{\mu r^{N-1}} \right\}$$

Hence in the Markov process of Model 2, either predators or prey are certain to become extinct in finite time. This result is also true for Model I, this can be shown using the same value of u_j but with slightly different condition on the constants. It is also true if constant immigration terms are added to the birth rates of both prey and predators.

EXTINCTION PROBABILITIES IN SPECIAL CASES

It has already been mentioned that ultimate extinction of one population is certain, it considers the determination of the probability $\theta(P,H)$ that the predator population is the first to become extinct, where (P,H) is the initial position (P,H=0,1,.....). For extinction probabilities it is only necessary to consider the imbedded Markov chain, and this can be thought of as a two-dimensional random walk in the positive quadrant with absorbing

barriers along the axes. The walk is certain ultimately to hit one or other axis, and $\theta(P,H)$ is the probability that it hits the y-axis. This probability satisfies one of the following equations:

$$(\alpha PH + \beta P + \lambda H)\theta(P,H) = \alpha PH\theta(P+1,H-1) + \beta P\theta(P-1,H) + \lambda H\theta(P,H+1) \quad \text{Model I} \quad \cdots (4.5.11)$$

$$(\alpha PH + \beta P + \lambda H + \mu PH)\theta(P,H) = \alpha PH\theta(P+1,H) + \beta P\theta(P-1,H) + \lambda H\theta(P,H+1) + \mu PH\theta(P,H-1) \quad \text{Model 2} \quad \cdots (4.5.12)$$

For both models we have the boundary conditions

$$\theta(P,0) = 0\,, \quad \theta(0,H) = 1 \quad \text{for all } P, H = 1,2,\cdots \quad \cdots (4.5.13)$$

The quadratic terms make the solution of these equations intractable so in this section we shall consider special cases where λ , the prey birth rate is 0. These cases are intersting in their own right and also provide the basis for the approximate solutions to the general cases which are derived in the next section.

Model-I. $\lambda = 0$. When $\lambda = 0$, there are only two possible transitions: $(P,H) \rightarrow (P+1,H-1)$ with transition probability αPH or $(P,H) \rightarrow (P-1,H)$ with transition probability βP. This situation is identical to that of the general epidemic where P is the number of infectives and H the number of susceptibles. This problem has been extensively studied: see, for example, Bailey (1975). For our purposes, the factor P can now be cancelled from Equation (4.5.11), giving

$$(\alpha H + \beta)\theta(P,H) = \alpha H\theta(P+1,H-1) + \beta\theta(P-1,H).$$

This difference equation can easily be solved for successively increasing H to give

$$\theta(P,H) = \sum_{i=1}^{H} a_{Hi} \left(\frac{\beta}{\alpha i+\beta}\right)^{P} \quad (4.5.14)$$

where the coeficients a_{Hi} are determined by

$$a_{11} = 1,$$

$$a_{Hi} = \binom{H}{i}\left(\frac{\beta}{\alpha i+\beta}\right)^{H-i} a_{ii} \qquad H>i$$

and $\sum_{i=1}^{H} a_{Hi} = 1$.

Model 2. $\lambda = 0$: When $\lambda = 0$ in Model 2, P can be cancelled from (4.5.12) to give a second-order difference equation:

$$\alpha H\theta(P+2,H) - (\alpha H+\beta+\mu H)\theta(P+1,H) + \beta\theta(P,H) = -\mu H\theta(P+1, H-1).$$

Since $\theta(P,H)$ is a probability, the unique solution of this equation is given by

$$\theta(P,H) = \sum_{i=1}^{H} b_{Hi} n_{i2}^{P} \quad \ldots\ldots (4.5.15)$$

where $0 < n_{i2} < 1 < n_{i1}$ are the roots of

$$i\alpha n_i^2 - (i\alpha+\beta+i\mu)n_i + \beta = 0 \quad \ldots\ldots (4.5.16)$$

and the coefficients b_{Hi} are given by

$$b_{11} = 1 \ , \quad b_{Hi} = \left\{\frac{\mu i n_{i2}}{\beta(1-n_{i2})}\right\}^{H-i}\binom{H}{i} b_{ii}, \quad H>i \quad \ldots\ldots (4.5.17)$$

and

$$\sum_{i=1}^{H} b_{Hi} = 1 \ .$$

The probability that predators ultimately become extinct for both models for the special case when the prey birth rate is 0 has thus been obtained.

APPROXIMATIONS TO EXTINCTION PROBABILITIES IN THE GENERAL CASE

Model I. In order to derive an approximate solution to equation (4.5.11) it is first convenient to reparametrize the model by writing $\beta = \alpha N$, $\lambda = \alpha M = \alpha\rho N$. Here M,N are the numbers of predators and prey respectively at the deterministic equilibrium position, and $\rho = M/N$. In many practical ecological situation there are more prey than predators and hence $\rho < 1$. It shall, therefore, be assumed this to be the case here. Equation (4.5.11) can now be rewritten

$$(P,H/N+P+\rho H)\theta(P,H) = PH\theta(P+1,H-1)/N + P\theta(P-1,H) + \rho H\theta(P,H+1). \quad \text{------ (4.5.18)}$$

It is assumed that $\theta(P,H)$ has a power series expansion in ρ:

$$\theta(P,H) = \sum_{r=0}^{\infty} \rho^r \theta_r(P,H). \quad \text{--- (4.5.19)}$$

Irrespective of the convergence properties of (4.5.19), it may happen that the first few terms of the expansion provide a useful approximation to $\theta(P,H)$ at least for small ρ and a restricted region of the (x,y)-plane

When $\rho = 0$, and hence $\lambda = 0$, it has the simplified problem solved in this Section . and hence $\theta_0(P,H)$ is given

by (4.5.14). Since $\theta_0(P,H)$ satisfies the boundary conditions (4.5.13) it can be taken

$$\theta_r(P,0) = \theta_r(0,H) = 0 \quad \text{for all } P>0, H>0 \qquad (4.5.20)$$

The $\theta_r(P,H)$ can now be obtained by substituting (4.5.19) in (4.5.18) and equating coefficients of powers of ρ. This leads to the first-order difference equation in $\theta_r(P,H)$:

$$\left(1+\frac{H}{N}\right)\theta_r(P,H) - \theta_r(P-1,H) = H\theta_r(P+1,H-1)/N + H\{\theta_{r-1}(P,H+1) - \theta_{r-1}(P,H)\}/P$$

$$r = 1,2,\ldots,\quad P>0,\ H>0 \qquad (4.5.21)$$

Starting with r = 1, H = 1 and using (4.5.20) it gets:

$$\theta_1(P,1) = \sum_{i=1}^{P} \{\theta_0(i,2) - \theta_0(i,1)\} \Big/ \left\{ i\left(1+\frac{1}{N}\right)^{P-i+1} \right\}$$

The equations (4.5.21) with r = 1 can then be solved for successively increasing H to give

$$\theta_1(P,H) = H\sum_{i=1}^{P} \left[\theta_1(i+1,H-1)/N + \{\theta_0(i,H+1) - \theta_0(i,H)\}/i\right] \Big/ (1+H/N)^{P-i+1}$$

It can be shown that $\theta_1(P,H) \to 0$ as $P \to \infty$ for all fixed H. This is reasonable as it would be expected that prey would become extinct first in this case and hence that $\theta(P,H) \to 0$ as $P \to \infty$ for all fixed H.

Equations (4.5.21) can now be solved in a similar manner for successively increasing r and we obtain the general result:

$$\theta_r(P,H) = H\sum_{i=1}^{P} \left[\theta_r(i+1,H-1)/N + \{\theta_{r-1}(i,H+1) - \theta_{r-1}(i,H)\}/i\right] \Big/ (1+H/N)^{P-i+1}$$

for all $P, H, r = 1,2,\ldots$. Specific solutions can thus be derived for the $\{\theta_r\}$, but the expressions are

cumbersome: the sequential definitions given here are very convenient for computation.

The numerical values of the approximation will be discussed latter, they will be compared with upper and lower bounds on $\theta(P,H)$ determined by independent calculation.

Model 2. A similar method was used to try to obtain an approximate solution to (4.5.12). However in this case it gets a second-order linear difference equation for $\theta_1(P,H)$ and a general solution does not appear to be a practical proposition. When H = 1, the solution is given, following the method of Jordan (1960), by

$$\theta_1(P,1) = \frac{\mu\beta}{\alpha^2} b_{22} \left(n_{11}^P \left[\sum_{i=1}^{P} \left\{ (n_{12}/n_{11})^i - (n_{22}/n_{11})^i \right\} \Big/ i + \ln\left(\frac{n_{11}-n_{12}}{n_{11}-n_{22}} \right) \right] \right.$$
$$\left. - n_{12}^P \left[\sum_{i=1}^{P} \left\{ 1 - (n_{22}/n_{12})^i \right\} \Big/ i + \ln\left(\frac{n_{11}-n_{12}}{n_{11}-n_{22}} \right) \right] \right) \Big/ (n_{11}-n_{12}) \quad \cdots\cdots (4.5.22)$$

where n_{i1}, n_{i2} (i=1,2) are the roots of (4.5.16) and $b_{2,2}$ is given by (4.5.17). The numerical value of (4.5.22) is discussed at the last.

EXPECTED NUMBER OF STEPS TO EXTINCTION

The same method of approximation has been applied to estimate the mean number of steps before absorption occurs in the random walk for Model 1. This will give an indication of the stability of the predator-prey model as the mean number of changes will provide information on how rapidly extinction occurs

If d (P,H) is the expected number of steps to extinction for the initial position (P,H),

then
$$d(P,H) = 1 + \frac{\{\alpha PH\, d(P+1,H-1) + \beta P\, d(P-1,H) + \lambda H\, d(P,H+1)\}}{\alpha PH + \beta P + \lambda H} \quad \text{---(4.5.23)}$$

$P, H = 1, 2, \ldots$

where $d(P,0) = d(0,H) = 0$.

As before it is first considered $\lambda = 0$ and in this case it denotes the solution by $d_0(P,H)$: now

$$(\alpha H + \beta)\, d_0(P,H) = \alpha H + \beta + \alpha H\, d_0(P+1,H-1) + \beta d_0(P-1,H).$$

The solution is given by

$$d_0(P,H) = D_H + \sum_{i=1}^{H} d_{Hi} \left(\frac{\beta}{\alpha i + \beta}\right)^P \quad \text{----(4.5.24)}$$

where the constants D_H, d_{Hi} are given by

$$D_H = H + \frac{\beta}{\alpha} \sum_{i=1}^{H} \frac{1}{i},$$

$$\sum_{i=1}^{H} d_{Hi} = -D_H \text{ and } d_{Hi} = \binom{H}{i} \left(\frac{\beta}{\alpha i + \beta}\right)^{H-i} d_{ii} \quad H > i.$$

We now postulate the power series solution for $\lambda > 0$ as

$$d(P,H) = \sum_{r=0}^{\infty} \rho^r d_r(P,H) \quad \text{---(4.5.25)}$$

where, as before, $\rho = M/N = \lambda/\beta$. Substituting (4.5.29) in (4.5.23) and equating powers of ρ, the first term $d_0(P,H)$ in the expansion (4.5.25) is given by (4.5.24). Subsequent equations for $d_r(P,H)$ are almost identical to those for $\theta_r(P,H)$ in (4.5.21) and the solutions are as follows:

$$d_1(P,H) = H \sum_{i=1}^{P} \left\{ \frac{d_1(i+1,H-1)}{N} + \frac{1 + d_0(i,H+1) - d_0(i,H)}{i} \right\} \Big/ \left(1 + \frac{H}{N}\right)^{P-i+1}$$

$$d_r(P,H) = H \sum_{i=1}^{P} \left\{ d_r(i+1,H-1) + d_{r-1}(i,H+1) - d_{r-1}(i,H) \right\} \Big/ \left(1 + \frac{H}{N}\right)^{P-i+1}$$

for $r = 2, 3, \ldots$ and for all $P, H = 1, 2, \ldots$. The usefulness of this is given below in the numerical comparisons.

NUMERICAL COMPARISONS

In order to investigate the usefulness of the approximations developed in Section described above it is necessary to have some knowledge of the numerical values of $\theta(P,H)$. A method has been developed which gives lower and upper bounds for $\theta(P,H)$ and for small values of the parameter p these bounds were found to be very close. Absorbing barriers were introduced at $P = c + 1$ and $H = r + 1$ and the imbedded Markov chain of changes in the population size inside the finite rectangle $0 \leq P \leq c+1, 0 \leq H \leq r+1$ was studied. Absorption at one of the four straight-line boundaries is certain. If $\phi_1(P,H)$, $\phi_2(P,H)$ are the probabilities of absorption in the boundaries $P = 0$, $H = 0$ respectively for initial position (P,H) then

$$\phi_1(P,H) \leq \theta(P,H) \leq 1 - \phi_2(P,H); \qquad \text{------(4.5.26)}$$

this gives exact lower and upper bounds for , the probability that predators become extinct first. Both ϕ_1 and ϕ_2 satisfy (4.5.11) when Model I holds and (4.5.12) when Model 2 holds for $1 \leq P \leq c$, $1 \leq H \leq r$ with appropriate boundary conditions.

The solution involve the inversion of an rc X rc matrix, and by using a program designed to invert sparse matrices it was possible to obtain values for $rc \leq 500$. For small values of N and p it was found that the probabilities of hitting the outer boundaries were very small unless P or H were close to c or r respectively, so, for a range of values, $\theta(P,H)$ could be determined to within very narrow limits. For each set of parameter values chosen, different values of r and c (subject to $rc \leq 500$) could be used in order to minimize $1 - \phi_1(P,H) - \phi_2(P,H)$.

A typical set of results of Model 1 when the approximation (4.5.19) is very satisfactory is given in Table 1. It can be seen that the power series converges very rapidly, and indeed $\theta_0+\rho\theta_1$ gives a satisfactory approximation. This was the case for a range of parameter values with $\rho \leq \frac{1}{3}$. Except for (20,10) the bounded model was almost certain to hit one of the axes, and so the probability of predator extinction was known almost exactly. In all these cases the power series gave an excellent approximation. When the bounded model started at (20,10) there was a probability of 0.099 that it first hit the line P = 26. In this case one would expect the prey ultimately to become extinct. It is also worth noting that the probability varies very much with P and H; it is not sufficient to consider the probability of predator extinction only when the population starts at the deterministic equilibrium point.

When $P > \frac{1}{3}$ the power series converges less rapidly. For N = 12, p = $\frac{1}{2}$ it is necessary to include the first four terms though in this case the upper and lower bounds (4.5.26) are too far apart to verify the accuracy of the approximation. When $P > \frac{1}{2}$ it is necessary to include more than four tems of the power series to obtain apparent convergence.

The same technique was used to check the accuracy of the power series solution to the expected number of steps to extinction and the results were comparable to those for $\theta(P,H)$. Results for α = 1, β=6, λ=1 are given in

TABLE 1

$\alpha = 1, \beta = 6, \lambda = 1;\ N = 6, \rho = \frac{1}{6}$

P	H	$\sum_{r=0}^{m} \rho^r \theta_r(P,H)$ m=0	m=1	m=2	m=3	Lower bound ϕ	Upper bound $1-\phi_2$
5	5	0.7302	0.7559	0.7535	0.7527	0.7524	0.7524
10	5	0.4359	0.4697	0.4694	0.4690	0.4689	0.4689
20	5	0.1146	0.1311	0.1318	0.1318	0.1318	0.1318
5	10	0.6604	0.6673	0.6612	0.6596	0.6592	0.6593
10	10	0.3934	0.4170	0.4155	0.4151	0.4149	0.4149
20	10	0.1050	0.1189	0.1194	0.1194	0.1150	0.2140

TABLE 2

(P, H)	(5,5)	(10,5)	(20,5)	(5,10)	(10,10)	(20,10)
$\sum_{r=0}^{3} \rho^r d_r(P,H)$	13.49	17.09	19,11	25.85	27.75	28.72
Expected duration of finite process	13.54	17.11	19,11	25.89	27.27	27.34

Table 2. Except for the point (20,10) the approximation is excellent. As previously stated, in this case, the finite chain had a probability of 0.099 of hitting the boundary P = 26. Presumably this happens rapidly, so the expected duration of the finite model would be smaller than that

of the infinite model.

The comparatively small values of these expected duration suggest that usually extinction occurs fairly rapidly, without much oscillation, though with the deterministic equilibrium point at $(1,6)$, cycling about this point is impossible.

Although the power series expansion for Model 2 was not derived for $H>1$, the same technique was applied to test the usefulness of the approximation for $H=1$. It was found that $\theta_0+\rho\theta_1$ gave an excellent approximation to $\theta(P,1)$ when ρ was small.

CONCLUSIONS

The method derived gives excellent approximations to the probability of extinction of the predator population for Model 1 when both ρ and N are small. The results suggest that it should also give a good approximation to extinction probabilities when N is large. It has been shown that, for small N, the expected number of steps to extinction is quite small so there is not much evidence of repeated cycles about the equilibrium position. The probability that the predator population becomes extinct first depends very much on the initial sizes of the two populations.

It is hoped that this method of approximate power series solution will also be useful for obtaining approximate solutions to other non-linear stochastic processes.

REFERENCES

1. ADAMS, V D, DEANGELIS, D L & GOLDSTIN, R A (1980) STABILITY ANALYSIS OF THE TIME DELAY IN A HOST-PARASITOID MODEL. J. THEOR. BIOL 83, 43-62

2. BULMER, M G (1971) THE EFFECT OF SELECTION ON GENETIC, GENETIC VARIABILITY AMER NATUR 105, 201-211

3. BAILEY, N T J (1957) THE MATHEMATICAL THEORY OF EPIDEMICS, GRIFFIN, *LONDON*

4. _____ (1975) THE MATHEMATICAL THEORY OF INFECTIOUS DISEASES, 2ND EDN. GRIFFIN, LONDON

5. BUNOW, B and WEISS, G H (1979) HOW CHAOTIC IS CHAOS? CHAOTIC AND OTHER "NOISY" DYNAMICS IN THE FREQUENCY BOMAIN, MATH BIOSCI 47, 221-237

6. BARNTT, V D (1962) THE MONTE CARLO SOLUTION OF A COMPETING SPECIES PROBLEM. BIOMETRICS. 18, 76-103

7. BECKER, N G (1973) INTERACTIONS BETWEEN SPECIES:SOME COMPARISONS BETWEEN DETERMINISTIC AND STOCHASTIC MODELS. ROCKY MOUNTAIN J. MATH, 3, 53-68

8. BARTLETT, M S (1957) ON THEORETICAL MODELS FOR COMPETITIVE AND PREDATORY BIOLOGICAL SYSTEMS BIOMETRIKA 44, 27-42

9 BILLARD, L (1977) ON LOTKA - VOLTERRA PREDATOR PREY MODELS J, APPL. PROB. 14, 375-381

10. BLYTHE, S P, NISBET, R M, and GURNEY, W S C (1984) THE DYNAMICS OF POPULATION MODELS WITH DISTRIBUTED MATURATION PERIODS. THEO. POP. BIOL. 25, 289-311

11. _____	(1982)	INSTABILITY AND COMPLEX DYNAMIC BEHAVIOUR IN POPULATION MODELS WITH LONG TIME DELAYS. THEOR. POP. BIOL. 22, 147-176
12. CUSHING, J M	(1977)	INTEGRO DIFFERENTIAL EQUATIONS AND DELAY MODELS IN POPULATION DYNAMICS IN "LECTURE NOTES IN BIOMATHEMATICS". VOL 20 SPRINGER VERLAG, BERLIN.
13. _____	(1980)	MODEL STABILITY AND INSTABILITY IN AGE STRUCTURED POPULATIONS. J. THEOR. BIOL. 86, 709-730
14. _____	(1981)	STABILITY AND MATURATION PERIODS IN AGE STRUCTURED POPULATIONS IN "DIFFERENTIAL EQUATIONS AND APPLICATIONS IN ECOLOGY, EPIDEMICS AND POPULATIONS PROBLEMS" (S BUSENBERG AND K COOKE. Eds)PP. 163-181. ACADEMIC PRESS NEW YORK.
15. CUSHING, J M and SALEEM, M	(1982)	A PREDATOR - PREY MODEL AGE STRUCTURE, J. MATH. BIOL. 14, 231-250
16. CHIANG CHIN CONG	(1968)	INTRODUCTION TO STOCHASTIC PROCESSES IN BIOSTATISTICS. WILEY, NEW YORK
17 CARD, T C & KANNAN D	(1976)	ON A STOCHASTIC DIFFERENTIAL EQUATION MODELLING OF A PREDATOR -PREY EVOLUTION. J. APPL. PROB. 13, 429-443
18. DOOB, J L	(1953)	STOCHASTIC PROCESSES WILEY, NEW YORK

19. FISHER, R A (1918) THE CORRELATION BETWEEN RELATIVES ON THE SUPPOSITION OF MENDELIAN INHERITANCE TRANS. ROY. SOC EDINBURGH, 52, 399-433

20. _____ (1922) ON THE DOMINANCE RATIO PROC. ROY. SOC. EDINBURGH 42, 321-341

21. FISHER, J (1969) THE GENETICAL THEORY OF NATURAL SELECTION DERENDON PRESS, OXFORD

22. FRETWELL, S D (1972) "PUPULATIONS IN A SEASONAL ENVIRONMENT" PRINCETON UNIV. PRESS PRINCETON, N J

23. GRACE YANG & C L CHIANG (1982) ON INTER ARRIVAL TIMES IN SIMPLE STOCHASTIC APIDEMIC MODELS. J. APPL. PROB. 19, 835-841

24. GAZIS, D C, MONTROLL, E W and RYNIKER, J E (1973) AGE SPECIFIC DETERMINISTIC MODEL OF PREDATOR-PREY POPULATIONS: APPLICATION TO ISLE ROYALE IBM J. RES. DEV. 17, 47-53

25. GURTIN, M E & LEVINE, D S (1979) ON PREDATOR-PREY INTERACTIONS WITH PREDATION DEPENDENT ON AGE OF PREY. MATH BIOSCI 47, 207-219

26. GOEL, N S, MAITRA, S C and MONTROLL, E W (1971) ON THE VOLTERRA AND OTHER NON LINEAR MODELS OF INTERACTING POPULATIONS REV. MOD. PHYS. 43, 231-276

27. GURNEY, W S C, BLYTHE, S P and NISBET, R M (1980) NICHOLSON'S BLOWFLIES REVISITED NATURE (LONDON) 287, 17-21

28. HASSELL, M P (1978) "THE DYNAMICS OF ARTHROPOD PREDATOR-PREY SYSTEMS". PRINCETON UNIV. PRESS, PRINCETON, N J

29. HASELL, M P
LAWTON, J H and
MAY, R M (1976) PATTERNS OF DYNAMICAL BEHAVIOUR IN SINGLE-SPECIES POPULATIONS. J ANIM ECOL. 45, 471-486

30. HASTINGS, A (1983) AGE-DEPENDENT PREDATION IS NOT A SIMPLE PROCESS I. CONTINOUS TIME MODELS. THEOR. POP. BIOL. 23, 347-362

31. HARRIS, T E (1963) THE THEORY OF BRANCHING PROCESSES SPRINGER-VARLAG BERLIN

32. HOLGATE, P (1976) ON STOCHASTIC PREDATOR PREY MODELS. PROC 9TH INTERNAT BIOMETRIC CONF.II 351-365

33. JORDAH, C (1960) CALCULUS OF FINITE DIFFERENCES, 2ND EDN. CHELSEA, NEW YORK

34. KIMURA, M (1957) SOME PROBLEMS OF STOCHASTIC PROCESSES IN GENETICS, ANN. MATH STATIST. 28, 882-901

35. KARLINS, S (1966) A FIRST COURSE IN STOCHASTIC PROCESSES. ACADEMIC PRESS, NEW YORK.

36 KARLIN, S (1979a) MODELS OF MULTIFACTORIAL INHERITANCE I, MULTIVA-RIATE FORMULATIONS AND BASIC CONVERGENCE RESULTS. THEORET POP. BIOL. 15, 308-355

37. KEMPTHOARNE, O (1957) "AN INTRODUCTION TO GENETIC STATISTICS" WILEY, NEW YORK.

38. LOTKA, A J (1925) ELEMENTS OF PHYSICAL BIOLOGY WILLIAMS & WILKINS, BALTIMORE. M.d

39. LEVINS, R (1966) THE STRATEGY OF MODEL BUILDING IN POPULATION BIOLOGY AMER. SCI. 54, 421-443.

40. LECREN, E D KIPDING, C and MCCORMACK, J C (1977) A STUDY OF THE NUMBERS BIOMASS AND YEAR CLASS STRENGTHS OF PERCH (PERCA FLURIATILIS, L) IN WINDEMERE FROM 1941 TO 1966. J. ANIM. ECOL. 46, 281-307

41. MAY, R (1974) "STABILITY AND COMPLEXITY IN MODEL ECOSYSTEMS" 2ND ED. PRINCETON UNIVPRESS, PRINCETON N.J

42. McDONALD, N (1978) "TIME LAGS IN BIOLOGICAL MODELS". SPRINGER VERLAG NEW YORK

43. MAY, R M (1973a) "STABILITY AND COMPLEXITY IN MODEL ECOSYSTEMS". PRINCETON UNIV. PRESS PRINCETON, N J

44. MAYNARD SMITH, J (1974) "MODELS IN ECOLOGY". CAMBRIDGE UNIV. PRESS, LONDON.

45. MURDOCH, W W & OUTEN, A (1975) PREDATION AND POPULATION STABILITY ADV. ECOL. RET. 9, 2-31

46. _____ (1975) PREDATION & POPULATION STABILITY ADRAN ECOL. RES. 9, 1-125

47. NIELSEN, L (1980) EFFECT OF WELLEYE (STIZOSTEDION UITEUM UITREUM) PREDATION ON JUVENILE MORALITY AND RECRUITMENT OF YELLOW PERCH (PERCH FLAUESCENS) IN ONEIDA LAKE NEW YORK CAHAD. J. FISH.AQUAT SCI. 37, 11-19

48. NISBET, R M and GURNEY, W S C (1982) "MODELLING FLUCTUATING POPULATIONS" WILEY, NEW YORK

49. NUNNEY, L (1983) RESOURCE RECOVERY TIME. JUST HOW DESTABILIZING IS IT? IN "POPULATION BIOLOGY" H I FREEDMAN & C STROBECK. EDS. LECTURE NOTES IN BIOMATHEMATICS VOL. 52, PP 407-413, SPRINGER VERKY, BERLIN

50. _____ (1985a) THE EFFECT OF LONG TIME DELAYS IN PREDATOR PREY SYSTEMS THEOR. POP BIOL 27, 202-221

51. _____ (1985b) SHORT TIME DELAYS IN POPULATION MODELS. A ROLE IN ENHANCING STABILITY ECOLOGY, IN PRESS.

52. PIMM, S L (1982) "FOOD WEBS" CHAPMAN AND HALL LONDON

53. PAINE, R (1965) NATURAL HISTORY LIMITING FACTORS AND ENERGETICS OF THE OPISTHOBRANCH NAVANAX INERMIS ECOLOGY. 46, 603-619

54. PARZEN, E (1962) STOCHASTIC PROCESSES HOLDEN-DAY, SANSFRANCISCO

55. REUTER, G E H (1957) DENUMERABLE MARKOV PROCESSES AND THE ASSOCIATED CONTRACTION SEMI-GROUPS ON I. ACTA MATH. 97, 1-46

56. _____ (1961) COMPETITION PROCESSES PROC 4TH BERKELEY SYMP. MATH STATIST. PROB. 3, 421-430

57. SMITH, R H & MEAD, R (1974) AGE STRUCTURE & STABILITY IN MODELS OF PREDATOR-PREY SYSTEMS. THEOR. POP. BIOL. 6, 308-322

58. _____ (1979) ON PREDICTING EXINCTION IN SIMPLE PUPULATION MODELS I. STOCHASTIC LINEARIZATION J. THEORET BIOL. 80, 189-203

59. _____ (1980) ON PREDICTING EXTINCTION IN SIMPLE POPULATION MODELS II NUMERICAL APPROXIMATIONS J. THEORET. BIOL. 82, 525-535

60. SCHAFFER, H E (1970) SURVIVAL OF MUTANT GENES AS A BRANCHING PROCESS IN: MATHEMATICAL TOPICS IN POPULATION GENETICS (ED. K KOJIMA) SPRINGERVERLAG, BERLIN

61. VOLTERRA, V (1926) VARIAZOINI EFFUTUAZIONI DEL NUMERO DINDIVIDUI IN SPECIE ANIMALI CONVIVENTI MEM. ACAD LINCEI ROMA 2, 31-113

62. WRIGHT, S (1921) SYSTEMS OF MATING III. ASSORTATIVE MATING BASED ON SOMATIC RESEMBLANCE GENETICS 6, 144-161

63. WANGERSKY, P J and CUNNINGHAM, W J (1957) TIME LAG IN PREDATOR-PREY POPULATION MODELS ECOLOGY 38, 136-139

R E F E R E N C E S

1. ADAMS, V D DEANGELIS, D L & GOLDSTIN, R A (1980) STABILITY ANALYSIS OF THE TIME DELAY IN A HOST-PARASITOID MODEL. J. THEOR. BIOL 83, 43-62

2. BULMER, M G (1971) THE EFFECT OF SELECTION ON GENETIC, GENETIC VARIABILITY AMER NATUR 105, 201-211

3. BAILEY, N T J (1957) THE MATHEMATICAL THEORY OF EPIDEMICS, GRIFFIN, LONDON

4. _____ (1975) THE MATHEMATICAL THEORY OF INFECTIOUS DISEASES, 2ND EDN. GRIFFIN, LONDON

5. BUNOW, B and WEISS, G H (1979) HOW CHAOTIC IS CHAOS? CHAOTIC AND OTHER "NOISY" DYNAMICS IN THE FREQUENCY BOMAIN, MATH BIOSCI 47, 221-237

6. BARNTT, V D (1962) THE MONTE CARLO SOLUTION OF A COMPETING SPECIES PROBLEM. BIOMETRICS. 18, 76-103

7. BECKER, N G (1973) INTERACTIONS BETWEEN SPECIES:SOME COMPARISONS BETWEEN DETERMINISTIC AND STOCHASTIC MODELS. ROCKY MOUNTAIN J. MATH, 3, 53-68

8. BARTLETT, M S (1957) ON THEORETICAL MODELS FOR COMPETITIVE AND PREDATORY BIOLOGICAL SYSTEMS BIOMETRIKA 44, 27-42

9 BILLARD, L (1977) ON LOTKA - VOLTERRA PREDATOR PREY MODELS J, APPL. PROB. 14, 375-381

10. BLYTHE, S P, NISBET, R M, and GURNEY, W S C (1984) THE DYNAMICS OF POPULATION MODELS WITH DISTRIBUTED MATURATION PERIODS. THEO. POP. BIOL. 25, 289-311

11. _____ (1982) INSTABILITY AND COMPLEX DYNAMIC BEHAVIOUR IN POPULATION MODELS WITH LONG TIME DELAYS. THEOR. POP. BIOL. 22, 147-176

12. CUSHING, J M (1977) INTEGRO DIFFERENTIAL EQUATIONS AND DELAY MODELS IN POPULATION DYNAMICS IN "LECTURE NOTES IN BIOMATHEMATICS". VOL 20 SPRINGER VERLAG, BERLIN.

13. _____ (1980) MODEL STABILITY AND INSTABILITY IN AGE STRUCTURED POPULATIONS. J. THEOR. BIOL. 86, 709-730

14. _____ (1981) STABILITY AND MATURATION PERIODS IN AGE STRUCTURED POPULATIONS IN "DIFFERENTIAL EQUATIONS AND APPLICATIONS IN ECOLOGY, EPIDEMICS AND POPULATIONS PROBLEMS" (S BUSENBERG AND K COOKE. Eds)PP. 163-181. ACADEMIC PRESS NEW YORK.

15. CUSHING, J M and SALEEM, M (1982) A PREDATOR - PREY MODEL AGE STRUCTURE, J. MATH. BIOL. 14, 231-250

16. CHIANG CHIN CONG (1968) INTRODUCTION TO STOCHASTIC PROCESSES IN BIOSTATISTICS. WILEY, NEW YORK

17 CARD, T C & KANNAN D (1976) ON A STOCHASTIC DIFFERENTIAL EQUATION MODELLING OF A PREDATOR -PREY EVOLUTION. J. APPL. PROB. 13, 429-443

18. DOOB, J L (1953) STOCHASTIC PROCESSES WILEY, NEW YORK

19. FISHER, R A (1918) THE CORRELATION BETWEEN RELATIVES ON THE SUPPOSITION OF MENDELIAN INHERITANCE TRANS. ROY. SOC EDINBURGH, 52, 399-433

20. _____ (1922) ON THE DOMINANCE RATIO PROC. ROY. SOC. EDINBURGH 42, 321-341

21. FISHER, J (1969) THE GENETICAL THEORY OF NATURAL SELECTION DERENDON PRESS, OXFORD

22. FRETWELL, S D (1972) "PUPULATIONS IN A SEASONAL ENVIRONMENT" PRINCETON UNIV. PRESS PRINCETON, N J

23. GRACE YANG & C L CHIANG (1982) ON INTER ARRIVAL TIMES IN SIMPLE STOCHASTIC APIDEMIC MODELS. J. APPL. PROB. 19, 835-841

24. GAZIS, D C, MONTROLL, E W and RYNIKER, J E (1973) AGE SPECIFIC DETERMINISTIC MODEL OF PREDATOR-PREY POPULATIONS: APPLICATION TO ISLE ROYALE IBM J. RES. DEV. 17, 47-53

25. GURTIN, M E & LEVINE, D S (1979) ON PREDATOR-PREY INTERACTIONS WITH PREDATION DEPENDENT ON AGE OF PREY. MATH BIOSCI 47, 207-219

26. GOEL, N S, MAITRA, S C and MONTROLL, E W (1971) ON THE VOLTERRA AND OTHER NON LINEAR MODELS OF INTERACTING POPULATIONS REV. MOD. PHYS. 43, 231-276

27. GURNEY, W S C, BLYTHE, S P and NISBET, R M (1980) NICHOLSON'S BLOWFLIES REVISITED NATURE (LONDON) 287, 17-21

28. HASSELL, M P (1978) "THE DYNAMICS OF ARTHROPOD PREDATOR-PREY SYSTEMS". PRINCETON UNIV. PRESS, PRINCETON, N J

29. HASELL, M P LAWTON, J H and MAY, R M (1976) PATTERNS OF DYNAMICAL BEHAVIOUR IN SINGLE-SPECIES POPULATIONS. J ANIM ECOL. 45, 471-486

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31. HARRIS, T E (1963) THE THEORY OF BRANCHING PROCESSES SPRINGER-VARLAG BERLIN

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36 KARLIN, S (1979a) MODELS OF MULTIFACTORIAL INHERITANCE I, MULTIVA-RIATE FORMULATIONS AND BASIC CONVERGENCE RESULTS. THEORET POP. BIOL. 15, 308-355

37. KEMPTHOARNE, O (1957) "AN INTRODUCTION TO GENETIC STATISTICS" WILEY, NEW YORK.

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56. _____ (1961) COMPETITION PROCESSES PROC 4TH BERKELEY SYMP. MATH STATIST. PROB. 3, 421-430

57. SMITH, R H & MEAD, R (1974) AGE STRUCTURE & STABILITY IN MODELS OF PREDATOR-PREY SYSTEMS. THEOR. POP. BIOL. 6, 308-322

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60. SCHAFFER, H E (1970) SURVIVAL OF MUTANT GENES AS A BRANCHING PROCESS IN: MATHEMATICAL TOPICS IN POPULATION GENETICS (ED. K KOJIMA) SPRINGERVERLAG, BERLIN

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63. WANGERSKY, P J and CUNNINGHAM, W J (1957) TIME LAG IN PREDATOR-PREY POPULATION MODELS ECOLOGY 38, 136-139